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Table of Contents

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ORIGINAL ARTICLES— PAGE.

"Types of Angina Pectoris", by M. D. SILBERBERG, M.D. .. 35
"Syphilis of the Heart and Great Vessels", by GUY LENDON, M.D., M.R.C.P. .. 41
"Cardio-Vascular Syphilis as Revealed at Post Mortem Examinations", by J. BURTON CLELAND, M.D., Ch.M. .. 46
"Prostatectomy with Closure of the Bladder, with Reference to a Modification of the Harris Operation", by W. JOHN CLOSE, M.B., M.S., F.R.C.S.E., F.R.A.C.S. .. 47

REPORTS OF CASES—

"Senile Tuberculous Arthritis", by H. C. RUTHERFORD DARLING, M.D., M.S., F.R.C.S., F.R.F.P.S. .. 51
"Acute Disseminated Encephalomyelitis with Recovery", by S. DEVENISH-MEARES, M.B. .. 52
"An Open Safety Pin in the Œsophagus of an Infant Forty-Seven Days Old Removed by Endoscopy", by RAYMOND HENNESSY, M.B., F.R.C.S. .. 53

REVIEWS—

Diphtheria .. 54

LEADING ARTICLES—

The Medical Practitioner and Mental Disorder 55

CURRENT COMMENT—

The Surgical Infections .. 56

ABSTRACTS FROM CURRENT MEDICAL LITERATURE— PAGE.

Pædiatrics .. 58
Orthopædics .. 59

BRITISH MEDICAL ASSOCIATION NEWS—

Scientific .. 60
Nominations and Elections .. 62

CORRESPONDENCE—

Erythrœdema .. 62
Anthrax .. 63
The Workers' Compensation Act .. 63
Physics .. 63
"Avertin" .. 63

PROCEEDINGS OF THE AUSTRALIAN MEDICAL BOARDS—

Victoria .. 64

BOOKS RECEIVED .. 64

MEDICAL APPOINTMENTS VACANT, ETC. .. 64

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 64

EDITORIAL NOTICES .. 64

TYPES OF ANGINA PECTORIS.[1]

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Remarks on the History of Angina Pectoris.

THE condition known as *angina pectoris* presents many problems for solution. Definition is difficult; classification unsatisfactory; the origin of the pain is obscure. Prognosis is uncertain, and the treatment is often far from gratifying.

Heberden's accurate description in 1768 leaves little to be added to the clinical picture. He conferred the name *angina pectoris* on the condition to indicate a strangling oppression in the chest, and today we are no further than a simple definition of "paroxysmal pain in the chest in relation to cardiac disease".(1)

[1] Read at a meeting of the Victorian Branch of the British Medical Association on May 4, 1932.

Jenner, very soon after Heberden's time, associated coronary disease and angina, and in this present era Parkinson and Bedford(2) go so far as to state that "the study of coronary thrombosis has nailed angina forever to the tree of decaying coronary arteries". If, however, we are to label all varieties of substernal or precordial pain in relation to cardiac disorders as *angina pectoris*, we shall have to include those which are quite harmless to life, and those of all grades of severity, culminating in the most desperate and dreaded coronary thrombosis.

The modern tendency is to recognize no division into *angina pectoris* and pseudo-angina, or into *angina minor* and *major*. Certainly coronary thrombosis is to appear as a separate entity, but every other form of anginal pain is to be regarded as *angina pectoris*. Paul D. White(3) criticizes the attempt of even such men as Mackenzie and Osler "to separate so-called secondary or pseudo-angina

from primary or true angina (as being) an impossible and unwise step".

There are logical grounds for abandoning the term pseudo-angina. As Potain remarked in 1894, "there are no false diseases; there are only false diagnoses". But, on practical grounds, there are very great advantages in separating groups which are not dangerous to life, however much they diminish efficiency, from those which are hazardous, potentially and actually. The use of the qualifying terms, "minor" and "major", scarcely convey the distinction, for "minor" may be merely an early stage of "major" angina, and in any case, the possibility of sudden death is not in any way related to the degree of pain.

Personally, I think that Mackenzie's division of anginal cases into primary and secondary has very great advantages. It conveys the thought that in the former group we are dealing with a basis of cardio-vascular disease fraught with very real danger, and in the latter group, with a background of a hypersensitive nervous system, a heightened receptivity to painful stimuli, and a low threshold for pain. Such cases demand an investigation for causes extracardiac, for psycho-toxic states, mental stress and strain, anxiety and fatigue neurosis, focal infection, social, physical and psychical maladjustments. There is no danger of sudden *exitus* in these cases. Surely it is obvious that some such differentiation is imperative.

In the past seven to ten years angina due to coronary thrombosis has been clearly demarcated as an entity that can be accurately diagnosed *ante mortem*. This advance has certainly cleared up some of the obscurity of anginal problems.

Classification.

I would like to put forward, provisionally, the following classification of paroxysmal anterior thoracic pain related to cardio-vascular disorders.

Angina Pectoris.

A. Cardio-Aortic Angina Pectoris.
- (i) *Angina pectoris* (Heberden, 1768).
- (ii) Coronary thrombosis.
- (iii) Aortitis.
- (iv) Ruptured heart.

B. Neurogenic Angina Pectoris.
- (i) Vasomotor angina (Nothnagel, 1867).
- (ii) Pseudo-angina (J. Latham, 1812).
- (iii) Reflex angina (Huchard, 1899).
- (iv) Secondary angina (Mackenzie, 1923).

C. Mixed Types.
Combinations of *A* and *B*.

Theories of Causation.

The mechanism producing anginal pain, its distribution and radiation, are matters of great interest; but the theories of causation can be only briefly referred to here.

Heberden gave seven reasons for regarding *angina pectoris* as a spasmodic disorder as opposed to inflammation. Jenner, a contemporary of his, was so confident of the association with disease of the coronary arteries that he won a wager on it.

Allan Burns (1809) attributed the condition to ischæmia and asphyxia of the heart muscle from disproportion between the amount of blood which the heart muscle needed and the amount of blood supplied to it. He used the comparison with pain produced in exercised muscles, when a ligature is tied round a limb. Potain in 1890 used the term "intermittent claudication of the heart" to express a similar idea.

Lewis[4] has recently reinvestigated this problem of pain produced in exercised limb muscles when the blood supply is obstructed. His conclusions are that the pain is due to accumulation in the tissue spaces of a chemical or physico-chemical substance, provisionally called "factor *P*". This is not due to oxygen lack, but probably accumulates owing to deficiency in the rate of diffusion and dispersion, depending on the obstruction to the blood supply. The application of these experiments to anginal pain is significant, the obstruction to the circulation being due in some cases to coronary spasm, in others to occlusion by thrombosis or embolism. In the latter, diffusion of "factor *P*" would be more delayed, hence the prolonged bout of pain.

Allbutt (1915) regarded the aorta as the seat of pain, due to distension of the diseased artery and irritation of hypersensitive endings in inflamed areas in the periaortic tissues. Sudden death he held to be due to depressor vagal inhibition. Wenkebach and Vaquez support this view.

W. D. Reid (1924) elaborates a similar idea and regards the attack as a failure of the depressor mechanism to lighten the load of the heart and aorta.

Mackenzie's views (1923) are well known, and rightly so. Few writers have made such painstaking, thorough and thoughtful clinical investigations. He regards the heart muscle as the source of the pain. The distribution he attributes to viscero-sensory and viscero-motor reflexes, along the sympathetic afferents to the corresponding spinal cord segments, mainly eighth cervical and first to fourth dorsal, and thence to the peripheral skin and muscle sensory fields.

Pain may radiate along the other segments and other related pathways, which explains unusual distribution of the anginal pain, for example, up the neck, over the jaws, or to the epigastrium. This explanation he gives for primary *angina pectoris*; in secondary cases the pain is produced because of undue sensitivity of the receptor side, so that even normal stimuli may be sufficiently summated to produced pain, where the threshold for pain perception is very low, as is commonly found in patients with anxiety neurosis.

Danielopolu reverts to the idea of relative coronary insufficiency for the myocardial demands. Also fatigue products irritate the sensory nerves and cause, not only pain, but reflex pressor stimulation.

East and Bain (1931) express a similar idea of sudden coronary insufficiency, or a disproportionate demand with insufficient supply.

K. F. Wenkebach insists that the pain is due to arterial stasis and distension of arterial lumen, the arteries being the seat of pathological distension. The relief of stasis is brought about by the opening of the peripheral circulation, either through the depressor reflex or by remedies such as nitrites. Warmth, also heart failure, may bring about a similar result by lowering peripheral resistance.

Can we harmonize these varying views? The factors concerned are the heart muscle, coronary arteries and blood flow, together with the nerve terminals and pathways. Unquestionably all are concerned. The coronary vessels, blood supply and myocardium form one unit and nerve afferents are the link to the conscious perception of pain. Sudden dramatic death is most likely due to ventricular fibrillation. Heart muscle is apparently insensitive to ordinary pain stimuli. The sensitivity of arteries has been much less investigated, but we know that acute pain may be experienced when the radial artery is punctured for samples of blood, and that the sympathetic nerves are intimately related to arteries.

Distension, spasm or occlusion of coronary arteries may be a source of pain, apart from the effects produced in the heart muscle by ischæmia, œdema and anoxæmia. On the other hand, some such substance as the hypothetical "factor *P*", postulated by Lewis, may accumulate in the tissue spaces to provoke the pain stimuli when the blood supply is interfered with.

Under experimental observation the heart muscle as a whole cannot go into a state of cramp or spasm. This would be incompatible with survival. But are we so certain that a few fibres could not go into painful spasm? The clinical picture, however, does not suggest that the pain is felt in the heart itself, but that it is best explained on Head's and Mackenzie's views, namely, as a viscero-sensory and viscero-motor reflex related to the appropriate skin and muscle segments (usually from the eighth cervical to the first four dorsal).

Neurogenic angina (if we are entitled to call it by the name *angina pectoris*, and I agree with those who think we are so entitled) is due to hypersensitivity of the receptor elements of the nervous system. Gross disease of the heart and arteries is not necessarily a factor in the production of the attacks.

It should be quite obvious that combinations and varying ratios of functional and organic causes may frequently occur; and clinical experience abundantly bears out this contention.

The aortic origin of angina applies to some cases, but is not a satisfactory explanation of the majority. The objections to this theory are that aortic disease is not necessarily found *post mortem*. It does not easily explain cases with low blood pressure or coronary thrombosis attacks. Vagal inhibition is unlikely to be fatal.

In the present state of knowledge it does not seem advisable to advance theories more dogmatic than those expressed above.

Clinical Types.

I now propose to consider the three principal types of angina which emerge from clinical investigation, namely, *angina pectoris*, neurogenic angina and the angina of coronary thrombosis.

Classical Angina Pectoris and Its Varieties.

It is unnecessary to elaborate the clinical picture of *angina pectoris*; it is too well known and has been often vividly portrayed in medical writings. The chief exciting causes are exertion, meals, flatulent distension, cold and excitement. The diagnosis is based far more on a painstaking history and evaluation of all the subjective phenomena than on physical examination. It is usual to find definite evidence of cardio-vascular disease, such as arteriosclerosis, high blood pressure, enlarged heart, valvular disease, arrhythmias, as well as electrocardiographic and radiological evidence. On the other hand, physical examination may reveal nothing abnormal—the arteries not sclerosed, blood pressure normal or low, and the size, sounds, rate and rhythm of the heart normal. Supplementary investigation by electrocardiograph or X rays may also reveal no abnormality, and yet the case is indubitably *angina pectoris*. It is rare, however, to find entirely normal electrocardiograms, though there is no typical graph, such as may appear in coronary thrombosis.

It is unusual for the medical practitioner to see the patient in an attack, and so he is dependent on the description obtained from the patient, relatives or friends. The circumstances of the first attack are specially enlightening; the form and degree of exertion often give a clue. The relation to meals is most deceptive to patient and physician. The commonest diagnostic error is to regard anginal pain as indigestion, because the pain or tightness in the chest occurs after meals and is associated with flatulence. The patient is naturally likely to rationalize, and so attributes the pain to something which is not serious. Investigation shows that it is meals *plus* exertion that initiate the attack. Frequently it occurs in a man over forty-five years of age, proceeding to work after a meal. He becomes conscious of substernal oppression, which soon amounts to pain. This is sufficiently severe to bring him to a halt. Relief comes, frequently with the accompaniment of belching. Commonly, in an attempt to repeat the relief, he may acquire the habit of aerophagy, or air swallowing, and then considers himself a martyr to indigestion.

The degree of pain and its radiation are very inconstant factors. The pain may be of all degrees, ranging from mild and bearable to intolerable and agonizing. The degree of apprehension depends on three factors—the temperament of the patient, the actual amount of pain, and the amount of fear implanted by the medical adviser. There may be no radiation of pain at all. Radiation is usually associated with the severer degrees only. The typical distribution is to the sensory areas associated with the eight cervical and first four dorsal spinal seg-

ments, that is, the inner border of the left arm, and the precordium. Radiation to the adjacent segments and associated nerves frequently occurs. Thus the pain may spread up to the throat, over the jaws, so as to resemble severe toothache; to the ears, and occasionally to the back of the head. It may spread to any distance down the arm, for example, to the shoulder, the elbow, the wrist and the fourth and fifth fingers. Sometimes it traverses the posterior aspect of the arm and the thumb.

Radiation down both arms for varying distances is not at all unusual. It may also penetrate to the scapular region, and sometimes toward the left hypochondrium and epigastrium.

The anginal nature of such aberrant pain is determined by its association with exertion or its origin in the substernal or precordial area.

Some patients describe a reversed spread of pain, commencing in the arm and spreading thence to the chest; or only pain in the arm, yet due to angina. Occasionally radiation is to the right side of the chest, or down the right arm, and not the left at all. In the instances which have come under my notice there was no clinical or radiological evidence of displacement of heart or aorta.

In a few cases pain first appears in the interscapular region, radiating through to the front of the chest. One patient, whilst walking, suffered from pain at the base of the neck, which then spread over the upper part of the chest, "as if it were in the wind-pipe and the top of the lungs".

In some patients the pain commences at the beginning of a certain effort, such as the first hole at golf. It then goes away, and the whole round can be played without further pain. No doubt the mental perturbation and stage fright at the onset of the game, stimulating the sympathetic nervous system and adrenals, are the factors in this rather unexpected effect.

Some patients play bowls with ease, yet cannot walk a few hundred yards. Here again there may be the psychical factor of interest, which allows of more harmonious interplay of the nervous reactions and also distracts from the conscious perception of afferent stimuli. Thalamic sensations may not be translated to cortical appreciation.

In some instances the patient complains of tightness and oppression in the neck and chest rather than pain, though commonly both are associated. There is usually a graphic description of the attack; but this largely depends on the temperament of the patient and his power of expression. The dull, stolid, unresponsive individual may give quite an inadequate account of his attack, whilst a highly strung or cultured individual may over-emphasize the picture. The physician should listen and observe patiently, in order to evaluate temperament, and thus form an opinion as to whether the description conforms to angina or to neurosis.

A distinction may be drawn, I think, between a graphic and a vivid description. Vice-like grip, crushing or clutching oppression, agonizing pain, with the characteristic spread, is graphic. John Hunter stated that it felt "as though the sternum were drawn back to the spine", whilst Matthew Arnold experienced a sensation "as though a mountain were upon my chest". A vivid description, such as "a spray of hot paint over the chest", certainly suggests neurosis. I am also unimpressed by the description of a sudden momentary knife-like stab. This is practically always part of a neurogenic angina.

Pain is the most important element in the picture; but on coming to a conclusion one should deliberate on the whole clinical gamut of signs and symptoms. It is often of great assistance to see electrocardiograms, and also radiograms of the heart and thoracic aorta.

Gall-bladder disease is a frequent association. This is not unexpected when one considers the age group common to both these conditions. In forty-two cases suspected of having a syphilitic basis, the Wassermann reaction was positive in only eight.

Space will not permit of elaboration of every detail in the diagnosis of *angina pectoris*. One point may be added. In a series of three hundred and ninety of my patients, over 20% were distinctly obese types. Obese people who are heavy smokers are particularly prone to *angina pectoris*.

Neurogenic Angina.

The second group in the classification of *angina pectoris* is represented by neurogenic angina. I mention it with due apology for introducing still another synonym in the large collection of terms applied to this functional variety of angina.

It is a common disorder in females under fifty years of age. One should be very chary of diagnosing *angina pectoris* in young females in the absence of organic cardio-vascular disease. In such, true angina is almost unknown. The functional variety is also frequent in young males, aged from twenty to forty years, particularly as part of the effort syndrome of returned soldiers. It is not at all infrequent in medico-legal cases, and in "the doctor-made neurasthenic", brought about by the fear implanted by a loose diagnosis of a "leaking valve" or "strained heart".

The associated symptoms are those of neurasthenia, of anxiety and fatigue neurosis. Consequently there are numerous subjective symptoms of a psycho-toxic nature. Undue fatigue is rarely absent; the patients are always tired and lack vigour. Chronic fatigue nearly always depends on chronic toxæmia, and a careful survey should always be made to discover focal or general infection. Mild quiescent tuberculous toxæmia may be present. Recurring rheumatism and toxic goître should be considered. Intestinal stasis may also be a cause. Palpitation and pain may result from the excessive use of tobacco, alcohol and tea, but rarely coffee in this country. Recent infections such as influenza, tonsillitis, pneumonia *et cetera* often cause prolonged debility and neurosis, which may become associated with precordial aching pain.

Psychical influences have numerous origins, based on childhood repressions and conflicts, mental stress

and strain, anxiety states arising from domestic, marital, social, sexual and financial maladjustments, and in the various phobias.

Patients suffering from neurogenic angina are unduly introspective, owing to fear and apprehension of heart disease. Strain and fatigue from nursing a sick relative is a common history.

The patient with *angina pectoris* feels particularly well between his spasms of pain; not so the neurogenics. They never feel well, and have a host of vague symptoms, vaguely localized, and at the same time vividly described with a wealth of detail. For fear that some be forgotten, out comes the little sheet of notes and memoranda. Similarly, the pain is vaguely located, not substernal or precordial, but usually infra-mammary and towards the left axilla. It may be compared to a neuralgic ache or to a sudden momentary stab or a hot burning sensation. It comes at any time, and for no particular reason, just as often when the patient is at rest or in bed as on exertion. It may be especially induced by fatigue, worry or excitement.

Hypotonic, visceroptotic, hypersensitive individuals are the principal sufferers. They often present the so-called asthenic habitus, being of long, narrow, thin build, with evidence of faulty somatic visceral and vasomotor tone. It should be fully realized, of course, that these unfortunate people are not immune from organic disease. The subjective symptoms so predominate that these patients are assumed frequently to be wholly neurotic, and the organic basis is overlooked.

If we visualize the physiological response to psychological factors, then these cases show up in clearer perspective. We should realize that the symptoms are based on disturbance of endocrine and sympathetic reactions. Thus are explained the associated signs of sweating, tremors, tachycardia, flushes, faintness, giddiness, variable blood pressure *et cetera*.

Some of these patients present very great difficulties in correct classification. It is a big responsibility to label a patient as having *angina pectoris*, with all the dread import of such a term. It is equally so to have the moral courage to assure patients that they have not true angina and that they need not fear sudden death. Errors are bound to be made in both directions; but I do not believe that it is a satisfactory position to play merely for safety and infallibility by labelling all cases *angina pectoris*. It is expected of us to take the responsibility to make the correct diagnosis and to share the satisfaction of placing the case in the right category. The mental comfort of the patient is naturally very great if he can be assured that his case is of neurogenic origin and that there is no fear of disaster.

Some of these patients complain of breathlessness. It is not a true dyspnœa, but an urgent desire for occasional long, sighing, deep breaths.

Hyperalgesia of the precordial area is also commonly present. An unduly long history, without evidence of progression, makes one suspicious that there cannot be a basis of coronary disease.

The following is a fairly typical example:

A.C.C., a female, aged fifty-three years, seen in 1929, complained of intermittent pain about the heart for ten years. The past history included double nephropexy, performed twenty years previously. She had had four children, one only surviving. Two had died under tragic circumstances, one had died in infancy. Normal menopause had occurred four years previously. The pain resembled dull toothache, and she felt a desire to hold her hand over the cardiac area. The pain appeared when she was lying on the left side or when she rose in the morning, and was not especially associated with exertion. She also complained of headaches and flatulent distension, not related to meals.

Examination showed her to be of a thin atonic type. The heart was not enlarged and the sounds were normal. The systolic blood pressure was 140 and the diastolic 80 millimetres of mercury. The pulse rate was 96 per minute. The thyreoid gland was not enlarged. The urine was clear. Both kidneys were easily palpable, being in a low position and freely movable. I regarded her as suffering from neurogenic angina associated with visceroptosis and hypotonicity.

Coronary Thrombosis.

Our third type of angina is that due to thrombosis or sudden occlusion by embolism of the coronary arteries. Because of the severity of the attacks and the high mortality it is perhaps the most important of all. It is a form that interests the surgeon, as it may very closely simulate the acute surgical emergencies of the upper part of the abdomen. Attention has particularly centred on this syndrome in the last ten to fifteen years, largely as a result of American work in correlating *post mortem* evidence of myocardial infarction with a history of unusually severe and prolonged angina.

J. B. Herrick described coronary thrombosis as an entity in 1912; but very little interest was taken until his next paper in 1918.

British interest dates from 1925. Pardee in 1920 described electrocardiographic changes in clinical cases comparable with those obtained experimentally after ligation of the left coronary artery. These important changes have been thoroughly investigated by Parkinson and Bedford in 1928, and by others.

Angina due to coronary thrombosis is characterized by being exceptionally severe and very prolonged, and by being accompanied by serious shock. The age incidence is much the same, or earlier than in *angina pectoris*, and males predominate by about five to one.

Previous mild anginal attacks may have occurred, or the attack may appear as a first paroxysm. It may be precipitated by exertion, but quite commonly occurs during rest in bed, during sleep, or after a heavy meal. The severity of the attack depends upon the size of the coronary branch involved and the rapidity of the occlusion. Prognosis is also affected by these factors, as well as by recurrence. The extent of arterial anastomosis is important.

The chances of survival are about 50% for a single attack. The well defined and easily recog-

nizable attack commences with substernal oppression and pain. Instead of passing off in a minute or so, or being relieved by belching, or by taking a hot drink, or using a vaso-dilator such as amyl nitrite or trinitrin, it rapidly becomes terrifically severe, with unendurable agony. The patient at first is immobile, but failing to get relief, becomes restless, thrashes about in the bed, gets on to the floor, clutches at his chest or throat, and gasps out for relief. Sweating freely, retching, belching or vomiting, and breathless with slightest movement, the victim is in unmistakable misery. The face is ashen grey and anxious, the eyes sunken or staring, the lips livid. A peculiar leaden blueness of the lips and finger nails indicates failure of the peripheral circulation and is an ominous sign. The heart sounds are feeble and the pulse is weak and of poor volume. The rate is variable and the rhythm may be irregular, usually on account of extrasystoles. The blood pressure falls conspicuously, usually from 60 to 80 millimetres of mercury, and may be registered with difficulty.

The pain may remain substernal or spread across the chest, into the arms, up to the throat, or at times to the epigastrium. The whole attack may last for half an hour, many hours, even two days or more. It is only relieved by large doses of morphine and leaves the patient very exhausted. The temperature rises to about 37·8° to 38·3° C. (100° to 101° F.) and may persist for some days. Leucocytosis of about 15,000 to 20,000 cells per cubic millimetre may occur.

In a fair number of cases a pericardial friction rub may be heard over the sternal region and sometimes over a wider area. It may be transient only or may persist for a week. In Levine's[5] series of 145 cases, pericarditis was noted in 13·8%. Frequently fine crepitations due to œdema of the lungs appear at the bases.

There are many variations in this typical syndrome. A first attack may be fatal, without evidence of previous angina or heart disease, or it may appear after years of ordinary angina. In some cases severe dyspnœa and shock without pain have been recorded, with typical *post mortem* findings. All cases are not so desperately severe as indicated above. If a small branch only be involved, the patient may be fairly well next day. Many patients have attributed previous mild angina to indigestion.

When there is acute prolonged pain about the level of the diaphragm, associated with shock, pyrexia, leucocytosis, vomiting or retching, there is obvious close similarity to abdominal emergency. Acute gastritis, perforated gastric or duodenal ulcer, acute cholecystitis, ruptured gall-bladder, acute pancreatitis and acute appendicitis, all have to be considered in the diagnosis.

A carefully taken history and a knowledge of all details leading up to the attack are essential. A story of pain and flatulence after meals is not enough. One really needs to know if this means pain after exertion following a meal. Radiation of pain down the arms is not a feature of abdominal lesions, though pain may be referred to the point of the shoulder in disease in the upper part of the abdomen. Very real difficulty in diagnosis occurs when it is known that the patient has had a previous attack of gall-stone colic.

In the examination of the patient special attention should be paid to a condition of feebleness of heart sounds and very low blood pressure. This may be deceptive when the fall, though great, is relative only (for example 200 to 130 millimetres of mercury), particularly when the previous pressure is not known. The presence of a pericardial rub clinches the diagnosis. Arrhythmia of some kind is quite frequent, and pulmonary crepitations point to a cardiac lesion rather than abdominal.

Electrocardiograms taken on a portable apparatus are decidedly helpful, but not necessarily diagnostic, during or quite soon after the attack. Characteristic changes tend to appear within a week or less. It should not need emphasis that transporting a patient should not be risked in order to secure electrocardiograms. However, electrocardiograms may clinch the diagnosis and save the patient from an unnecessary operation. A portable instrument can be used without disturbing the patient.

In considering a diagnosis of acute abdominal conditions, the important point is in remembering to investigate for coronary thrombosis and not merely concentrate attention on the abdomen.

The age incidence and sex incidence are of some importance. *Angina pectoris* is very rare in young females and comparatively rare in young males in the absence of definite heart disease, such as mitral stenosis or aortic disease. A half-hourly temperature and pulse chart is valuable, particularly as an aid to the detection of peritonitis. Apart from those affected with biliary and renal colic, persons suffering from abdominal disease are not restless. They instinctively remain quiet in an attempt to localize the inflammation. Vomiting is not so marked in coronary cases, when it consists more of retching and eructation; nor is abdominal rigidity such a feature. A persistent ashen colour of the face is more typical of a cardiac condition, except in relation to the immediate shock of a perforated viscus.

The practitioner must weigh a number of small points and use all his acumen in deciding whether the pain and symptoms are of abdominal or thoracic origin.

Comparatively few patients suffering from *angina pectoris* or from the effects of coronary thrombosis are admitted to public hospitals. Only the more urgent and severe are taken to the wards. As would be expected, the mortality in the hospital cases is very high. The following is a typical example.

G.H., a male, aged seventy-two years, admitted on February 15, 1931, died on August 28, 1931. For twelve months prior to admission he had suffered from pain in the chest whilst walking up hill. In the latter three months the pain would radiate to the left arm and was severe enough to cause him to stop in order to obtain relief. The pain would then recur with further walking. At bedtime, fourteen days before admission, he had suffered from a sudden attack of pain in the front of the chest, shooting down the left arm to the hand. The pain was described

as "severe and tearing", causing him to roll about and become very breathless. It had lasted for one and a half hours. Since then it had recurred almost daily and he had become very weak and breathless. The patient was of ashen appearance and his lips were cyanotic. The pulse was irregular from extrasystoles. The systolic blood pressure was 105 and the diastolic 90 millimetres of mercury. The apex beat was about 11·25 centimetres (four and a half inches) from the mid-line. The area of cardiac dulness extended for 1·25 centimetres (half an inch) lateral to the right border of the sternum. There were a few crepitations at the bases of the lungs. He improved slightly, but remained feeble and had mild recurrences of anginal pain. On July 15, 1931, he suffered an attack of acute pulmonary œdema. He died on August 28, 1931, just over four months from the onset of coronary thrombosis.

At *post mortem* examination the weight of the heart was found to be 480 grammes (sixteen ounces). The aorta was enlarged and atheromatous patches were present. In the right coronary artery there were atheromatous plaques which were brittle and almost occluded the lumen. Mural thrombosis had occurred in the apex of the right ventricle, and there was a small infarct at the apical wall. There were also pericardial adhesions over the left side of the ventricle.

The principles of treatment in coronary thrombosis are as follows.

A period of rest in bed, depending on the severity of the symptoms and signs, is necessary. Usually a minimum period of six weeks is desirable. Pain should be relieved by injections of morphine. Very careful nursing, avoiding undue movement in the early stages, is necessary. Digitalis should be used with discretion, and mainly for congestive failure. Stimulants may be occasionally required, for example, "Coramine", "Cardiazol", strychnine. Sedatives may be necessary if there are much restlessness and apprehension, for example, "Luminal" and bromides. Coronary dilators may be used, though the action is uncertain, for example, theobromine and theophylline compounds. Diet should be small in amount and with liberal glucose.

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SYPHILIS OF THE HEART AND GREAT VESSELS.[1]

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History.

THE results of syphilitic infection first began to be observed about the year 1500, and in 1542 the French physician Fernelius first suggested that syphilis might be the cause of aneurysm. His contemporary Vesalius wrote much more fully on both subjects, and is reputed to be the first person to diagnose aortic aneurysm, both abdominal and thoracic, in the living. However, Vesalius makes no mention of syphilis in connexion with aneurysm. It is significant that peripheral aneurysms, with their ætiology of trauma, alone were known to earlier investigators dating back to Galen, and that the aortic aneurysm with its arresting characteristics, both during life and after death, was unknown until after the syphilization of Europe.

Osler, who was rarely at fault with medical history, states that Ambroise Paré was the first to associate aneurysm with syphilis; but the majority of works of reference aver that Paré blamed the mercurial treatment of syphilis for its production, not the disease. One hundred and fifty years later we find Morgagni insisting on the syphilitic ætiology; but once again the suggestion was not accepted. Since Welch's statistics of aneurysm and aortitis, taken from the army record in 1876, the ætiology has never been seriously disputed, and in this century the Wassermann reaction and the discovery of the *Spirochæta pallida* in the aorta itself have put the matter beyond doubt.

As regards syphilis of the heart itself, the condition was suggested by Corvisart in the early nineteenth century; but the first definite pathological record of gummatous myocarditis was made by Ricord in 1845, and soon afterwards confirmed by Virchow and Sir Samuel Wilks.

Welch described a syphilitic aortitis in 1876, and in the nineties syphilitic sclerous myocarditis, syphilitic coronary disease and aortic valvular changes, due to the then unknown virus, were freely described.

More recent contributors to the study of cardiac syphilis are legion, the most important clinically being Osler and Allbutt, and pathologically Klotz and Warthin.

Incidence and Statistics.

Syphilis of the heart and great vessels is a comparatively rare disease, certainly in general practice, but in a general hospital, as with so many other disorders, cases are much more common. This is partly due to the disabling nature of the disease, and partly to the ready access to the laboratory and radiographic aids to diagnosis which are available in a clinic. But specialized methods, added to *post mortem* records, have been of great service to the clinician, in so far as he can now offer with some confidence a diagnosis that thirty years ago was rarely made until the disease had advanced beyond all hope of treatment.

Syphilis affects the heart and great vessels in one of four ways: aortitis, aortic insufficiency, aneurysm, myocarditis. The latter does not imply or include gumma, an excessively rare finding in these cases.

Every syphilitic is a potential candidate for syphilitic cardio-vascular disease, and a few statistics are necessary to indicate the proportion of syphilitics who develop this late manifestation. The figures I am showing may be somewhat vitiated, because the negro, by habit or ill-luck, appears especially likely to contract syphilis, and to

[1] Read at a meeting of the South Australian Branch of the British Medical Association on April 28, 1932.

avoid treatment, and to suffer from its later manifestations.

In an institution where the Wassermann test was applied to every patient, 7% of white and 25% of black patients reacted. In these syphilitic cases 14% of white and 33% of black patients gave definite evidence of aortic disease, that is, aneurysm or regurgitation.

The malevolent effects of syphilitic as compared with other forms of heart disease are well shown by the following figures: Mortality among sufferers from syphilitic heart disease, 38%; mortality among sufferers from non-syphilitic heart disease, 17%. Of 1,000 autopsies on white people, in 200 (20%) evidence of organic cardio-vascular disease was found, and in 25 (2½%) evidence of cardio-vascular syphilis, the *post mortem* incidence of the disease being twice as high as the clinical incidence. This is due in the first place to the number of bodies of persons who have died suddenly brought to the institution; secondly, to the fact that much cardio-vascular syphilis is subclinical in degree, and possibly to the liability, which still exists, in the *post mortem* room, to label as syphilitic lesions those which really are degenerative. Also of organic heart lesions, the percentage of syphilitic lesions varies from 5% to 15%, according to the admixture of negroes in the population from which the figures are taken.

In the following statement is shown the field covered by syphilis in connexion with cardio-vascular disease.

Cardiac or Vascular Disorder.	Percentage Syphilitic.
Aortic aneurysm	75%
Abdominal aneurysm .	35%
Aortic regurgitation ..	30%
Ventricular failure ...	20%
Heart block	10%
Auricular failure	2%
Cardiac pain	1-3%

The most striking feature of the distribution of the lesions is their overwhelming prevalence in the ascending and transverse portions of the aorta. Proceeding towards the heart the disease stops abruptly at the aortic valves, and in the opposite direction rapidly fades in the descending aorta. This is well exemplified by Turnbull's dictum that aortic syphilis is four times as common in the ascending aorta as in the abdominal aorta, and also by the fact that over three-quarters of all aneurysms in the area of origin of the great vessels are syphilitic, but only one-third of those of the abdominal aorta have that same ætiology. The pulmonary artery escapes, and the reason is not clear; the lower intrapulmonary pressure would doubtless save it from aneurysm were its walls infected, but only rarely have examples of syphilitic pulmonary arteritis been noted. These unusual cases exhibit Ayerza's syndrome, namely, polycythæmia, cyanosis, and enlargement of the spleen; Leonard Rogers has described a sclerosis of the pulmonary artery, with a similar symptom complex, amongst young women in southern India, which he thinks is syphilitic.

Syphilis, apart from the primary lesion, attacks the tissues of the body in three successive phases, both in the congenital and in the acquired infections: Bacteriæmia, or secondary stage; focal, or tertiary stage (for example, the gumma); lymphogenous, or quaternary, or parasyphilitic stage. Cardio-vascular syphilis belongs essentially to the quaternary stage in conjunction with tabes and general paralysis of the insane, and it is in all probability a lymph-borne virus which is responsible for those lesions. Deaths from aortic regurgitation and aneurysm six months after primary infection are reported, and I have seen aortic regurgitation develop within two months of an extragenital chancre. However, these cases are of such extreme infrequency that they need not divert attention from the fact that the latent period is very constantly in the vicinity of twenty to twenty-five years, rising at times to forty years as an extreme. This period is actually longer than the average latent period of neuro-syphilis, but the discrepancy can probably be accounted for by considering that the central nervous system is a more sensitive tissue, affording symptoms more rapidly and consequently bringing the patient to seek relief before the cardio-vascular system, which, when it fails, does so relatively suddenly. There is a very similar sex correspondence also, 90% of the patients being males; but this is offset by the venereal disease clinic figures, which all show a five to one male preponderance of new syphilitic infections. However, in congenital syphilis of the aorta the sexes are equal, and in this form nearly all aortic and nervous disease shows signs of its presence before twenty years of age, which is possibly an expression of the more intense nature of the infection.

Before going any further into the very debatable question of the pathogeny of these lesions I propose to consider the clinical aspect and see what questions the clinician may fairly ask the pathologist to solve.

The Clinical Aspect.

The Patient's History and Symptoms.

One of the most constant features of the history in these cases is that the patient is completely unaware of his disorder until subjective symptoms appear. A history of syphilitic infection is common, and a history of treatment, very rarely of thorough treatment, is occasionally obtained, and according to some investigators a positive Wassermann reaction is found in 95% of all cases. Other figures in the history concern polyarthritis, which occurs in 10% of cases, in only one-third of which, however, is mitral stenosis found.

The actual onset is relatively abrupt, the patient's attention frequently being drawn to his cardiac inefficiency by his inability to perform some task requiring slightly more effort than usual. Early

subjective sensations may be indefinite and frequently include the following: weakness, giddiness, fainting, smothering sensation, and cough. These symptoms are closely followed by dyspnœa, pain and dropsy.

In aortic insufficiency the symptoms almost always appear in the order given above, but when aneurysm exists, alone or combined, four-fifths of the patients commence their definite history with pain.

Pain.—Paroxysmal pain may follow effort in syphilitic or any other organic cardiac disease, but paroxysmal pain without effort, especially that which wakes the patient from his sleep, is a striking feature of syphilitic infection. Even more pathognomonic is paroxysmal dyspnœa without effort; it has been estimated that in rheumatic carditis only 2% of patients suffer in this way, while in sphilitic regurgitation it is by far the most prominent symptom.

The pain is typically anginal in character, but it is the angina of decubitus rather than the angina of effort, and its abrupt paroxysmal features serve to separate it from coronary occlusion. As there is a real tendency to confuse the two, it may not be out of place to quote Levine, one of the early workers on coronary occlusion; he lays down two essentials for the diagnosis of infarction: (i) A period of severe and persistent precordial pain; (ii) the addition of new physical signs indicative of a change in the cardiac wall. He adds that only 3% of his patients suffering from coronary occlusion react to the Wassermann test. Carey Coombs confirms the rarity of the associated conditions by reporting only one case of coronary thrombosis amongst a series of one hundred cardiac syphilitics. In other words, in syphilis we expect, and we find, pain without sequelæ.

Physical Examination.

The signs most frequently found on physical examination are cardiac hypertrophy, increase of retrosternal dulness, and alteration of the aortic second sound, all of which will be separately considered.

Cardiac Hypertrophy.—Cardiac hypertrophy is very constant in the aortic group and conspicuously absent in the aneurysmal group. When the heart is enlarged to the left without a demonstrable cause, such as high blood pressure or valvular disease, with or even without a positive Wassermann reaction, syphilitic myocarditis should be suspected.

Retrosternal Dulness.—Clinically, retrosternal dulness is demonstrable in half the non-aneurysmal cases, but the percentage of actual aortic dilatation cannot be estimated unless all the patients are skiagraphed, for by this means alone can small degrees of dilatation be accurately determined.

Alteration of the Second Aortic Sound.—Accentuation of the second aortic sound usually means increased systemic blood pressure, but when the sound is qualitatively altered to resemble the booming of a native drum, the diagnosis of structural aortic change with or without high blood pressure can be confidently made; this occurs in degenerative as well as syphilitic aortic change. The French cardiologists are very keen on this point, and call the particular sound *bruit de tabourka*, after the Algerian tomtom.

Diminution in the aortic second sound, especially its loss in the vessels of the neck, is associated with aortic insufficiency.

The Murmurs.—The regurgitant murmur, indicative of insufficiency, is soft and conducted down the sternum. Many celebrated physicians have commented on this murmur, and some of their views may be worth recording here. Hoppe-Seyler was the first to state that the aortic diastolic murmur was best heard to the left of the sternum in rheumatism and to the right in syphilis, and he attributed this to the associated aortic dilatation in syphilis; but Strickland Goodall, who confirms this finding, states that the common occurrence of a spread of disease from the mitral valve to the posterior aortic cusp in rheumatism is the true explanation. Others aver that a diastolic murmur, heard only at the lower end of the sternum, is most often syphilitic in origin, and this has been my experience. This murmur may be inaudible unless listened for with a wooden stethoscope or by direct auscultation of ear to chest; possibly this is the earliest auscultatory finding. An appearance and disappearance of this early murmur has been noted by the French authors, but Horder is convinced that once the murmur comes, it remains and alters very little, any sudden increase in the murmur being due to coincident valvular rupture.

Mitral disease being such an uncommon accompaniment of syphilitic aortic reflux, the frequent occurrence (over 80%) of Flint's mitral presystolic murmur in this connexion is of interest. Many suggestions as to the nature of this murmur have been offered, but the most favoured is the impingement of the reflux stream on the mitral flaps causing an artificial stenosis. The presence or absence of a thrill was thought to distinguish between organic and functional stenosis, but so effective is this reflux stream that in 10% of cases there is an unmistakable apical presystolic thrill. Diagnosis of mitral disease under these circumstances is unjustified, unless all criteria of syphilitic infection are absent and definite rheumatic infection with signs of previous rheumatic carditis are present.

Disturbances of Rhythm.—Disturbances of rhythm (apart from *pulsus alternans*) are rare; of Carey Coombs's 100 patients none were affected with auricular fibrillation. Of 500 cases of fibrillation, aortic syphilis was present in only 3%. Premature contractions are more frequent.

Heart block is occasionally caused by a gumma of the bundle, but much more rarely than is generally realized; in fact, less than one-tenth of all patients suffering from heart block react to the Wassermann test. Contrast this with a series of rheumatic cardiac cases. In 85% of the fatal cases fibrillation occurred. One common fallacy should

be mentioned here, namely, the relationship of variation of the brachial blood pressure in the two arms; this occurs just as frequently in atheroma of the aorta as it does in dilatation and aneurysm, and should be disregarded.

Discussion of the Findings in Syphilitic Disease.

In any case of cardio-vascular syphilis, therefore, the chances are two to one that the patient has aortic reflux, even that he has aortic dilatation, ten to one that he has a normal rhythm, ten to one against the presence of aneurysm, and three to one against the presence of other syphilitic manifestations.

The concurrence of tabes with aortic syphilis is varied by the inclusion of negro cases; but 10% is a fair estimate for a white population. Other syphilitic findings in order of frequency are the following: (i) General paralysis of the insane, (ii) internal ophthalmoplegia, (iii) choroiditis, (iv) optic atrophy, (v) focal cerebral lesions, (vi) chronic glossitis, (vii) gummata.

Generalities.

We are now in a position to consider some generalizations about cardio-vascular syphilis.

As regards age, the valvular cases fit in between the rheumatic carditis of the twenties and the arteriosclerotic valve deformation of senescence; thirty-five years of age may be taken as the dividing line between rheumatism and syphilis. I have noticed that the Adelaide Hospital patients have been rather older at onset, quite a large proportion of cases beginning in the fifties.

The consequences of the syphilitic processes are added to and accelerated by atheroma, hyperpiesia, diabetes, alcoholism and rheumatism. The first mentioned (atheroma) combines remarkably well with syphilis in two ways: (i) in the arterial wall, where syphilitic inflammation selects the media, and atheromatous degeneration the intima; (ii) in the coronary arteries, where syphilis closes the orifices in the sinuses of Valsalva, and atheroma blocks them in their course through the cardiac muscle.

Unquestionably hyperpiesia must help to convert an aortitis into an aneurysm; but I have never thought that the two conditions were frequently associated; the French, however, lay great stress on the point.

Statistics show that the supervention of rheumatism upon syphilis is serious, but the reverse is not so.

A damaged valve (whatever the cause) is a favoured site for the development of bacterial endocarditis; but the damage done by rheumatism or a congenital abnormality is far more likely to be followed by bacterial endocarditis than is that caused by syphilis. There may be a time factor in this, for the survival period of the two former is considerably longer than the last mentioned, thus greatly increasing the possibility of bacterial endocarditis.

A question of some practical interest is the association of valvular rupture with valve disease, especially syphilitic disease. Apart from tremendous violence, such as a vehicle passing over the chest, and compressing the aorta during diastole, it is certain that a normal valve rarely, if ever, ruptures; but when an accident, such as bumping down on the heels from a kerb, ruptures an aortic valve, syphilis should be suspected. Horder, in his Lumleian Lecture in 1926, gives a wonderfully illustrative case.

A strong policeman slipped whilst carrying a corpse downstairs. He noted a sudden pain in his chest, felt faint, but soon recovered. That night his wife got out of bed, ostensibly to turn the cat out, but the sound of purring was coming from the constable's chest. He was found to have a positive Wassermann reaction and aortic regurgitation.

This point is of some importance in medico-legal cases; a claim for damages due to valvular rupture is unlikely to be justified, unless associated with extreme direct violence.

Pathology.

Turning now to the pathology of this condition, the most constant and striking lesions are found in the proximal part of the aorta; until the Wassermann test came into general use these lesions were frequently confused with atheroma, and the percentage of syphilitic cases in the *post mortem* records of one Australian city in particular was exceptionally high.

Perhaps the distribution of the lesions is the most helpful feature; the disease stops more or less abruptly at the beginning of the descending aorta, and if present elsewhere (for example, in the abdominal aorta), appearing in the form of a localized ring or band. Longitudinal wrinkling is said to be a most characteristic finding, and pouches large and small, the latter associated with stellate scars, are more likely to be syphilitic than atheromatous. But the real difficulty lies in the constant association of the lesions of both conditions.

Histologically syphilis shows itself as an inflammatory change invading the media from the intima, and spreading along the course of the lymphatics accompanying the *vasa vasorum*. The intimal change is inflammatory; but the fibrosis which occurs here is probably to buttress the weakened media. Atheroma works in the opposite direction, beginning as a degeneration of the intima encroaching on the adjacent media.

Both processes break up the elastic fibres, syphilis far more savagely than atheroma: long fissures form in the media, and the corresponding intimal thickening gives rise to the above-mentioned macroscopical appearances. This type of fissure is probably the basis of aneurysm formation. Though the abrupt cessation at the proximal end of the aorta spares the ventricular wall, in ending here it still strikes two vital points, the mouths of the coronary arteries and the aortic valve cusps, which are tied down by shortening and stiffening of the commissures; occasionally they become adherent. Incompetence may be caused without any valvular change by stretching of the aortic ring. Klotz first drew attention to this frequent occurrence and its significance. In support of this statement Professor Cleland tells

me that he has never seen aortic stenosis of syphilitic ætiology.

Concerning muscular lesions there is much variation of opinion. Carey Coombs thinks they are rare and difficult to unearth, being especially infrequent in the sino-auricular node and auriculo-ventricular bundle; but Warthin, who is a great authority on this particular point, constantly finds foci of round-celled infiltration "drowned" in serum, and muscular degeneration and replacement, and numerous spirochætes, which Carey Coombs had never been able to demonstrate, even with the aid of the leading French histologists. Possibly this discrepancy may be accounted for by the material examined, for after four centuries in Europe there must be a considerable inherited resistance to the disease as compared with the resistance of the African negro.

Pathological Explanation of Clinical Features.

Four points in the clinical section appear to require a pathological explanation and amplification.

1. Why is cardiac pain so common and cardiac infarction so rare?

2. How does syphilis prepare the endocardium for superimposed infections?

3. What is the nature of the myocardial lesion?

4. How is the long gap between the primary lesion and the onset of cardiac symptoms filled in?

All but the last can be fairly adequately answered.

Cardiac Pain.—There is no disease in the course of the coronary arteries themselves and no clotting within them, for it is the mouths alone which are affected. This can be beautifully demonstrated by the skiagraphy of injected specimens of syphilitic, as compared with arteriosclerotic, hearts. The pain is probably related to the obstruction of the coronary orifices, and possibly to periaortic inflammation. Pain can occur without any inflammation (as in pernicious anæmia), and is an expression of the failure of supply of oxygenated blood to the muscle cells; undue exercise is then abruptly checked by pain or sudden death.

Superimposed Infections.—In rheumatism the infective agent enters the depth of the valve from the coronary blood stream, excites a reaction which bursts the valve or floats off its endothelium, leaving a roughened surface; the vegetations are merely an index to the underlying disease. In syphilis also the process begins in the depths of the valve and causes it to swell, with the same effect on the endothelium, but the injury is inflicted, not from the coronary blood stream, but by a lymph-borne virus, filtering *viâ* adventitia and media to the commissure. Here the inflammation and consequent swelling interfere with the lymph flow, making the valve œdematous and fragile.

Myocardial Lesions.—Myocardial lesions have been discussed. To quote Carey Coombs: "The lesions are dystrophic, and whatever else may be laid to the charge of the spirochæte, it cannot be accused of remaining alive in the walls of the heart in sufficient numbers to excite obvious inflammatory response."

The Long Interval.—The question as to what happens in the interval between the time of the primary lesion and the onset of cardiac symptoms has given rise to endless discussion. The work and observations of Klotz appear to supply the most satisfactory answer.

> The inflammatory reaction is most intense in the connective tissue around the aorta. Prevented by the outer wall of the media from passing on in a straight line, it filters through by following the *vasa vasorum*. Consequently these vessels swell and cause medial splitting, the cracks becoming filled with inflammatory cells, organizing blood vessels, and new connective tissue. Lastly, the valves are deformed and the coronary mouths hampered.
>
> If some malign or purposeful agency were set to work, could it be more skilful at bringing the heart's work to an end?

Whence comes the virus? Can there be a common reservoir for these coeval aortic and spinal root lesions? In these cases and at this stage the lymph-borne lesion seems inevitable and the blood-borne phenomena (gumma) mere accidents. This compares with the central nervous system, where arteritis, myelitis and gumma are the exceptions, tabo-paresis the rule, at this late stage.

Of course, there are examples of overlapping, but they probably indicate a legacy left from the earlier stage, and do not invalidate the general truth of the thesis. Probably the lymph-borne virus provokes an inflammatory reaction in all the fibrous structures of the body, but it is the vital functions of the invested and compressed structures under discussion which give rise to such striking symptoms.

Klotz's three conclusions relating to lymph drainage are as follow:

1. The lymphatics of the aortic wall are unevenly disposed (compare the patchy infection of the abdominal aorta).

2. The aortic wall is richly supplied with nutrient vessels associated with liberal lymph drainage.

3. These lymphatics are in close and direct association with the larger lymphatic system surrounding the aorta as a whole, which has two large drainage beds, one in the thorax and one in the abdomen.

He notes the frequent concomitance of aneurysm with fibrous mediastinitis, and thinks the syphilitic virus comes from these beds to the aorta and central nervous system. This is the key to Babinski's syndrome (aneurysm and tabo-paresis) and removes all surprise at its occurrence. Many pathologists have noted and commented on the difficulty of delivering an aneurysm from its bed, and this fibrous mediastinitis may be the explanation.

Prognosis.

There can be no question that the grave prognosis of aortic regurgitation, laid down in all text books, is due in part to the exceptionally poor expectation of life of any sufferer from syphilis of the aorta. Most practitioners remember cases of aortic disease

of considerable duration. In this connexion I can quote the case of a man refused life insurance by Sir Joseph Verco forty years ago, and still alive; and again, a boy who won the half-mile race at his school sports at the age of sixteen. Both these persons suffer from aortic incompetence. In fact, I am inclined to believe that non-syphilitic aortic regurgitation has an outlook no worse than that of mitral regurgitation.

Duration of life in aortic syphilis depends on early diagnosis and treatment. Treatment is almost hopeless once heart failure has eventuated.

Treatment.

Wenn man nicht weiss wo wann warum
Dann gibt man jodkalium.

Uttered before the introduction of "Salvarsan", this aphorism still holds good. All are agreed that potassium iodide is the backbone of the treatment, supplemented by mercury and bismuth, but there is considerable difference of opinion as to the advisability of using arsenicals. Some think that they are essential, and use ordinary doses, but the majority use small doses and do not commence their use until after the heavy metals have been given a thorough trial. I have seen sudden death in three cases following "Novarsenobillon", and do not use organic arsenic unless the Wassermann reaction is obdurate to other treatment or no progress is being made. Obviously it is contraindicated in the presence of cardiac failure.

Results of Treatment.

Aneurysm.—When no treatment is given, the average duration of life from the date of diagnosis is said to be nine months; when one arsenical and one bismuth course is given, the duration is thirty-four months; and when more than one year's constant treatment is employed, sixty-nine months. (I feel sure this must include simple dilatation.)

Aortic Regurgitation.—When no treatment is given, 80% of patients die in less than two years; of those given one arsenical and one bismuth course, 75% lived for two years; those given efficient treatment, all lived two years.

Therefore, it is wise to treat the disease thoroughly and warn the patient that a diagnosis of aortic syphilis renders him unfit for strenuous exercise, strenuous work, or a strenuous indulgence in alcohol.

It will be interesting to see whether those who contracted syphilis in the late war and were treated early and efficiently, will progress to quaternary syphilis. I think it possible that some of those who were treated with "Salvarsan" compounds alone may eventually fall victims to parasyphilis in either its cardiac or nervous manifestations.

Acknowledgements.

Although I have made few direct references, I must acknowledge my indebtedness to the articles of Carey Coombs, Thomas Horder and Klotz, which have formed the basis of this summary.

CARDIO-VASCULAR SYPHILIS AS REVEALED AT POST MORTEM EXAMINATIONS.[1]

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A CAREFUL tabulation is kept of the lesions met with at *post mortem* examinations at the Adelaide Hospital, and the results are published in *The Medical and Scientific Archives of the Adelaide Hospital,* which appear annually. From these results, either already published or about to be published, the following tables indicate some of the information available on the subject of cardio-vascular syphilis as met with in patients dying at a public hospital in Australia.

TABLE I.
Cardio-vascular syphilitic lesions found post mortem in patients who had reacted and patients who had not reacted to the Wassermann Test.

	Wassermann Reaction Positive.	No Reaction to the Wassermann Test.
Numbers examined	30	111
Syphilitic aortitis extending to the aortic valves	7	1
Syphilitic aortitis resulting in aneurysm	5	1 (ruptured aneurysm)
Syphilitic aortitis alone	2	—
Total cases in which syphilitic disease was observed	14	2

In three cases also there were knobby aortic cusps, and in one, an aneurysm believed to be atheromatous. None of these reacted.

TABLE II.
Cardio-vascular syphilitic lesions in 2,400 post mortem examinations at the Adelaide Hospital.

Syphilitic aortitis with extension to the valve	19
Syphilitic aortitis with aneurysm	39
Syphilitic aortitis alone	28
Total	86 (3·5% of all cases)

Thus in nearly half the cases of syphilitic infection of the aorta aneurysm occurs, and in less than half of the remainder the disease reaches the aortic valve. Gumma of the heart causing heart block was found in one case.

For some years now a separate entry has been made of the pathological lesions in patients who have reacted and patients who have not reacted to the Wassermann test. In the small figures as yet available the tabulated results show that in nearly half of those patients with a positive Wassermann reaction syphilitic aortitis was present. It also shows that in two cases out of 111 in which there

[1] Read at a meeting of the South Australian Branch of the British Medical Association on April 28, 1932.

was no reaction to the Wassermann test, syphilitic aortitis was unquestionably present, that is, two of the patients were definitely syphilitic.

A second table shows the frequency of cardiovascular syphilitic lesions in 2,400 autopsies. The table speaks for itself and shows the percentage of bodies in which syphilitic aortitis was found *post mortem*. It will be seen that in about one-third of the cases in which syphilitic aortitis occurred, the disease had not extended to the valve or as yet caused an aneurysm. It is probable that, had these patients lived longer, some of them would have developed aneurysms. It is not, however, equally certain that, had the patient lived, there would also have been an extension of the syphilitic reaction in some cases to the aortic valve. I do not know whether there is any evidence available to show that all of the aorta that is going to be involved in the syphilitic reaction is involved at the beginning, that is, whether all parts of the aorta are invaded contemporaneously, or whether the disease over a period of years may extend either towards the cardiac side or towards the abdominal aorta. In syphilitic aortic disease we often find the process ceasing more or less abruptly a short distance above or a longer distance above the aortic valve. The question is: had these patients lived longer, would the reaction have approached still more closely to the valve, and eventually, after having surrounded the orifices of the coronary arteries, given rise to lesions in the cusps also?

Extension of the syphilitic reaction to the cusps of the valve leads to a retraction or rolling up of them, causing aortic regurgitation, but no definite obstruction to the passage of blood through the aortic orifice. In syphilitic disease the condition is a pure aortic regurgitation. In contrast to these appearances are those examples of knobby calcified aortic cusps which are rigid and which give rise to much stenosis. These have no relation to syphilis. The question is whether they represent atheromatous changes in cusps already injured by rheumatic fever and affected with rheumatic fibrosis, or whether the changes are purely degenerative, that is, atheromatous, or whether in some cases they may represent healing and calcification of vegetations in subacute bacterial endocarditis.

A good description of the above has been given by H. A. Christian (*The Journal of the American Medical Association*, July 18, 1931, page 158, quoted under the caption, "Epitome of Current Medical Literature" in *The British Medical Journal*, October 31, 1931, page 73). He seems to favour the view that these lesions are due to degenerative changes taking place in rheumatic valves. Dr. H. M. Turnbull, whose opinion was asked, inclines to consider these changes as primarily atheromatous, though the frequency of fusion of such rigid cusps suggests that there may be something more than pure degeneration, though he does not exclude pure degeneration alone. He does not think that the lesions are likely to be due to healed subacute malignant endocarditis.

PROSTATECTOMY WITH CLOSURE OF THE BLADDER, WITH REFERENCE TO A MODIFICATION OF THE HARRIS OPERATION.

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As an advocate of the suprapubic operation, with which this paper is mainly concerned, I shall defer consideration of the perineal methods of approach till the end, and then only to contrast similar steps that have evolved in the perfecting of either method.

A short *résumé*, submitted herewith, of the progress of prostatic surgery, which does not pretend to be anything like a complete bibliography, reminds one of the evolution of tonsillectomy, from blind enucleation or slicing to a precise dissection under vision, with the elimination, as far as is humanly possible, of all the risks. One also expects, as with the criticism of modern tonsillectomy, that some "die-hards" may object to the complicating of nineteenth century methods.

The acid test of the advantages of any innovation is, after all, the effect on the mortality and the morbidity. In studying the latter factor, statistics do not always give one the fairest idea of results, so that one falls back on one's own experience of immediate and remote effects. My mind is therefore still an open one on the question of the best method of performing prostatectomy, through lack of a sufficient number of cases to warrant dogmatism, though my personal observation of post-operative morbidity, and the statistics of mortality as supplied by others, give me the impression that progress up to modern closure methods has all tended towards improvement.

Blind Dissection and Hæmorrhage.

From the time of Freyer the main bogy has been hæmorrhage; in addition, sepsis and uræmia have threatened in the near distance. Up to a point, the power for evil of the latter spectres has been estimated beforehand and precautions taken to deal with them; but hæmorrhage has always been a lurking danger that might spring on one unawares. For this reason a reliable portal of retreat and attack has up till now been universally considered necessary; hence the large drainage tube. "As large as possible, sister," one has heard the surgeon say as he selected a three-quarter inch hose. And, indeed, such an outlet was a necessity for the ejection of clots which would otherwise have caused distension of the bladder and prostatic bed, with further hæmorrhage. In the mention of drainage I must be understood to refer to a tube placed in the wound of access, unless the urethral catheter is specially mentioned.

Preliminary closure, even without perfect control of bleeding, has, of course, been practised occasionally in apparently favourable cases. Ralph Thompson,[6] of Guy's Hospital, used to advocate it, though it appears from his most recent writing that drainage is now usually resorted to, and some

of us have seen our own A. M. Cudmore confidently do the same under some conditions.

Hæmostasis by Means of Compression Apparatus.

The methods of employing compression apparatus for the purpose of combating the tendency to bleed have in themselves prevented closure on account of the necessity of removing the apparatus. Among these may be mentioned the following.

1. The wrapping of gauze round the proximal end of a catheter in the urethra and applying traction on the distal end.

2. The packing of the cavity with gauze, either to push the superfluous mucosa into the cavity (Lower) to encourage its adherence, or packing after removal of the redundant mucosa.

3. The use of inflatable bags (Hagner, Pilcher, Ogier Ward *et cetera*), traction being applied externally, or, in the case of Ward's instrument, pressure maintained through the suprapubic wound and counter-pressure on the perineum by means of a clamp.

Hæmostasis without Apparatus, but with Drainage.

Methods of effecting hæmostasis without apparatus are the following.

1. Deep sutures are placed in the prostatic bed to obliterate the cavity in much the same way apparently as one would obliterate the cavity left by an enucleated uterine fibromyoma. Cabot has been credited with this questionable effort; but the latest edition of his book, at any rate, does not mention it; moreover, "thorough drainage" is his watchword.

2. There is a method employed by E. L. Keyes, Junior,[5] which, because of its anticipation of one of the principles of Harris's technique, will be described briefly. Keyes introduced a suture through the perineum by means of a Reverdin needle guided into the posterior part of the urethra by means of a staff. In this way he attempted to pick up the torn edge of the prostatic urethra, then, transfixing the posterior edge of the prostatic cavity from within outwards, the suture was again brought on to the perineum, and the two ends tied over a roll of gauze. More than one transfixion suture might be necessary to reconstruct satisfactorily the floor of the prostatic urethra. Drainage was provided by a suprapubic tube. "I do not care to do away with it altogether", said Keyes, and humorously referred to a surgeon who had recently (1914) advocated closure at a surgical congress, only to return to his own clinic and find that his method had been abandoned in his absence as an unsafe procedure.

Keyes's sutures were removed in eighteen to twenty-four hours, when primary hæmorrhage was unlikely to occur. Here, then, was apparently the first attempt to control arterial bleeding by traction of a tongue of mucosa over the posterior edge of the cavity, with also probably the secondary idea of reforming the floor of the prostatic urethra.

Other attempts have evidently been made to suture the internal sphincter to the torn remnant of the prostatic urethra, anterior to the verumontanum, according to J. B. Squier,[7] but "this", he adds, "is rarely possible". Presumably Young's boomerang needle and holder had not then been invented.

3. Thompson-Walker's[9] so-called "open operation", and similar methods of Judd, Hunt and others to control arterial bleeding by suturing the bladder margin of the cavity with separate or continuous sutures, have been a big step in the toilet of the cavity, but since they did not control venous oozing, they still necessitated the use of a drain.

Closure of the Bladder after Effecting Hæmostasis.

Finally, S. H. Harris[2][3][4] has evolved a method which, in his and other surgeon's hands, has allowed complete closure of the bladder and drainage by a urethral catheter. Briefly, his method aims, in addition to securing the main bleeders by separate ligatures, at drawing a wide-based tongue of mucosa forward and attaching it to the prostatic capsule in the neighbourhood of the torn urethral floor, thus reconstructing the floor of the prostatic urethra. The cavity is further narrowed anteriorly and superiorly and rendered more tubular by several transverse sutures. The retained catheter is given an extra eye and fixed by a long silkworm gut sling to the anterior abdominal wall. The silkworm gut sling can be cut for removal of the catheter on the tenth day.

After performing this operation myself I have observed some slight oozing into the bladder, unless my transverse sutures were drawn undesirably tightly round the catheter; I have, therefore, not felt justified in entirely discarding a small suprapubic drain. I now venture to suggest a modification of Harris's operation, which I have found simpler, and which has afforded in my hands a more certain hæmostasis, although it may seem a presumption on the part of one who has a mere half-dozen cases to prove its practicability, whereas the technique of Harris has gradually evolved into its present state of perfection by the performance of hundreds of operations.

A Modification of Harris's Operation.

My technique, then, in six recent cases, not consecutive ones, but by no means selected, has been as follows.

A vertical subumbilical incision, 7·5 centimetres (three inches) long (that is, sufficiently large to allow the introduction of the whole hand between the recti, as recommended by Thompson-Walker), and a bladder incision, sufficient only for the introduction of two fingers, are employed. The shelling out of the adenomatous mass is performed intraurethrally (see Figure I), so as to commence the enucleation on the deep aspect of the growth and preserve as much mucosa on the bladder aspect as possible. Tags and isolated small adenomata can then be removed under vision; but the mucosa is trimmed only if very thin and

redundant at any point. The object is to leave a wide fringe composed of mucosa, submucosa, and trigonal muscle overhanging the ledge of the cavity.

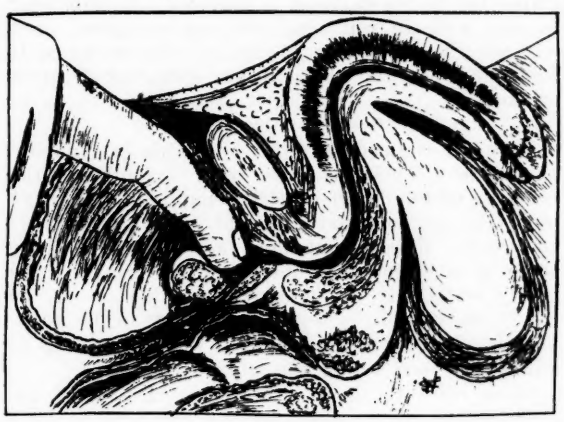

FIGURE I.
Showing the method of enucleating the prostate.

Bleeding, even from spurters, is ignored while a purse string is run round the fringe, as shown in Figure II, the field being kept as dry as possible by suction and tamponage. Applying traction on the anterior edge of the fringe, a running stitch of "number 2" plain catgut is passed in and out round the margin, transfixing it at six or seven spots. This suture is made possible by Harris's modification of the Young boomerang needle, holder and ligature carrier.

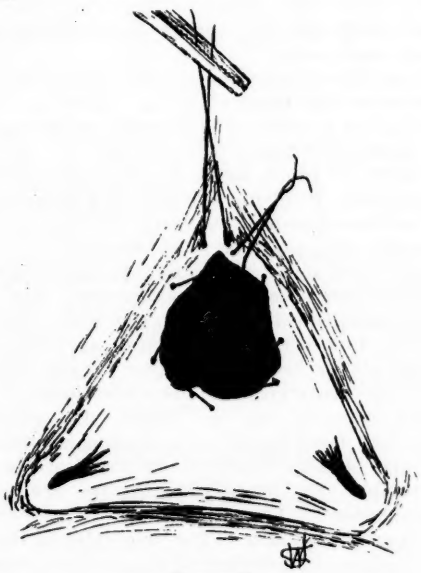

FIGURE II.
Showing the application of a purse-string suture to the fringe of mucosa.

A de Pezzer catheter, size "12 E", is now passed stretched on a curved introducer (see Figure IV). This instrument was made for me by Messrs. McNeil and Smith, of Adelaide. Traction on the catheter is relaxed when the expanded end projects into the bladder; the catheter is seized in the bladder, and the introducer withdrawn. The purse-string is now drawn tightly round the catheter and tied. Figure III depicts this stage of the operation, though the introducer which is here shown in position, would, of course, have been removed before the suture was tied.

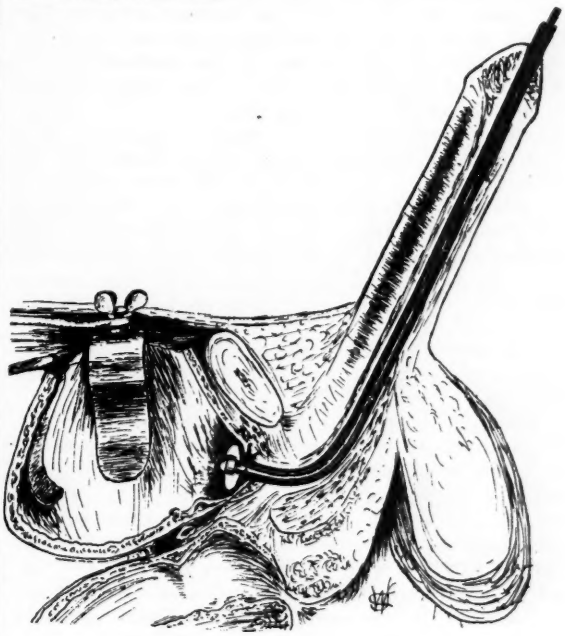

FIGURE III.
Showing the purse-string fastened round the catheter. (The introducer is here shown in position; it would have been withdrawn, of course, before the suture was tied.)

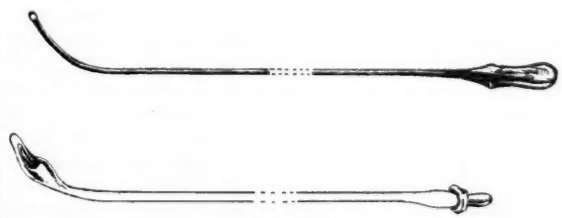

FIGURE IV.
Showing the introducer and the de Pezzer catheter with introducer fitted.

Traction is made on the distal end of the catheter sufficiently to pull the expanded end, together with a cone of mucosa, as far into the prostatic cavity as possible. The necessary amount of traction to secure perfect hæmostasis is easily gauged under vision, and is maintained by fixing the distal end of the catheter to one or other thigh by means of a strip of adhesive strapping. This fixation should be effected immediately by the nurse, to obviate dangerous blind traction during later stages of the operation. If any oozing into the bladder should be noted (though this is unusual), another stitch can be placed at any desired point so as further to tighten the hold on the catheter.

Any bleeding that may now occur from the prostatic bed can pass down the urethra alongside the catheter; it amounted in my cases to a few drops only, and is no doubt purely venous oozing, as the arterial bleeding has been controlled by the compression of the cavity edge by the cone of mucosa.

The above steps represent the essential differences from the method of Harris. Closure takes place by much the same plan as recommended by the latter, namely, by figure-of-eight sutures anchoring the bladder incision to that of the abdominal wall. I admit I was loath to do this at first, believing that such fixation disturbed the relations of the normal anatomy and would thus tend to interfere with function. On the other hand, it is probable that the ultimate fibrous attachment to the abdominal wall at this part does little more than take the place of the displaced urachus. At all events, the advantages of maintaining this weak spot in the bladder wall, well above the level of the meatus, justifies one in doing it. When I have not done so, leakage, from a few drops to a few fluid ounces has occurred into the pre-vesical space, without doing any harm, however, since safety drains had been provided from this space.

The pre-vesical space is not obliterated by me. My respect for it is very profound, and impels me to insert a roll of rubber tissue on each side of the bladder, projecting from the lower end of the wound. These are left in position till the fifth day, to guard against the retention of extravasated urine, should there be any escape from the bladder, for, unlike a sutured union in the intestine, of which the peritoneum proves such a useful ally, a perfectly watertight junction cannot be guaranteed.

It must be realized that the pre-vesical space of Retzius extends backwards on either side as far as the hypogastric vessels in most cases, though in some pelves only as far as the obturator foramen. An understanding of the allantoic sheath (that part of the endopelvic fascia which encloses the bladder) is necessary to appreciate this statement; I have described it more fully in an anatomical thesis shortly to be published. The postero-lateral prolongation of the pre-vesical space is named by French anatomists *l'espace pelvi-rectale supérieure*, the lowest point of which is considerably below the medial pubo-prostatic ligament. Extravasated urine will certainly stagnate here when only a single midline drain is employed; hence my two safety drains. J. B. MacAlpine[6] has pointed out that extravasation is a frequent cause of death supposed to be due to uræmia, and I myself am convinced that this was the cause of a profound toxæmia in several cases (not of this series) where there was not a commensurate rise in the blood urea content. H. B. Devine[1] recognizes this fact also in employing suction drainage to both the bladder and pre-vesical space.

In twenty-four hours traction on the catheter is relaxed by reapplying the strapping; by means of a long tube the urine is siphoned into a "Winchester" placed on the floor. The catheter is removed on the tenth day, though occasionally one may find it being forced out by bladder activity, as indicated by the passage of urine round it. I believe it is best for the suprapubic union, to allow at least a week to elapse before the catheter is removed. No difficulty is met with in removing this size catheter, even without the assistance of the introducer, since it folds up well, and the external sphincter is quite relaxed.

Results of Operation.

It is not claimed that this operation is a plastic one in the same sense as the term is applied to the covering of the defect in a cleft palate; its primary object is not to reconstruct the canal so much as to prevent bleeding, though there is undoubtedly some, if not complete, adhesion of the fringe to the cavity walls. It would depend on the size of the cavity, the amount of sepsis that occurs in the walls, and the viability of the cone that was inverted, as to whether a good lining was afforded.

I had the opportunity of doing a cystoscopy on the twenty-fourth day on a patient who was about to leave hospital, and, as far as I could see, the cavity was lined with healthy, though, naturally, somewhat œdematous mucous membrane. My object in doing the cystoscopy was to fulgurate some papillomatous buds which could not be reached before prostatectomy and which could not be identified with certainty when the bladder was open. Operation on this man had consisted in the resection of a border-line papilloma and the removal of two adenomatous masses from the prostate, each the size of a Barcelona nut.

In my first two cases there was some sloughing of the mucosal fringe, but I attributed this to a hemstitch purse-string suture which I first began to make use of. I have observed no sloughing since the attenuated parts, if any, of the fringe have been trimmed away and an oversew purse-string suture has been employed.

In three cases there was a short period of incontinence after the removal of the catheter, lasting from a day to a week; this was due to the use of a larger catheter, "number 16 E", and has not happened with the other cases, in which "number 12" has been used. I did not consider it a disadvantage at all, unless a wet bed, which up till then had been consistently dry, could be so considered.

In my short series there was no mortality, and the morbidity during convalescence has been minimal, excepting in one case, the fifth. This case could not, however, be considered an example of the risks of the technique. The patient was fortunately the best "risk" of the series to start with, but bled severely on the eighth day after the catheter had been forced out. It was necessary to reopen the bladder to clear out the clots, when a gauze tampon was found plugging the urethra. It is a confession of lamentable surgical carelessness rather than a weakness in the principles of the technique, that, in the presence of an excellent exposure and lighting, and a perfect hæmostasis, whereby the bladder was demonstrated to all present to be perfectly dry, it is possible to report such a calamity. There has, however, been no setback in this case since hæmorrhage was controlled. In future only spherical tampons with anchor tapes will be used in the bladder, and the final swabbing

"for luck" after the bladder margins have been allowed to collapse, must be dispensed with.

In one other case, complicated by vesical papillomata, mentioned above, a suprapubic drain was employed for about thirty-six hours on account of slight oozing from the resected area, but not from the prostatic cavity; after the removal of the drain progress continued according to plan.

Perineal Operations.

With regard to the perineal approach, whatever one's personal opinions may be of its disadvantages, one must admit that progress has been just as enterprising in the nature of packing and inflatable bags, and one operation at least for primary closure has been evolved, the technique of Wildbolz,[10] of Berne, being apparently safe as a method of controlling bleeding. Wildbolz removes enlarged lobes with their covering mucosa, secures a clean cut edge anteriorly and posteriorly, and brings these together in a transverse line of union by interrupted sutures.

Conclusion.

In the hands of different artists different methods of treating the same subject give equally satisfactory effects, and surgery is not an exception among the arts. In the operation of prostatectomy there are probably other methods that produce as good an immediate result as that which I have described above, but no method can be termed "sound" that does not include reasonable attempts to nullify the recognized risks.

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Reports of Cases.

SENILE TUBERCULOUS ARTHRITIS.[1]

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IN the second edition of "Clinical Lectures and Essays", by Sir James Paget, published over three-quarters of a century ago, is to be found a chapter on "senile scrofula". To the lucid clinical description of senile tuberculous arthritis contained therein, little has been added of late years. This essay draws attention to the fact that the old, that is, people of over fifty-five years of age, are more often tuberculous than those between thirty and fifty, and certainly are more often so than is generally supposed.

The distribution of senile tuberculous arthritis and its relative frequency at the different ages is shown by Alfer's table of statistics compiled from Trendelenburg's clinic at Bonn.

TABLE I.

The Numbers of Cases of Tuberculous Arthritis at Various Ages. (After Alfer.)

	40 to 45.	45 to 50.	50 to 55.	55 to 60.	60 to 65.	65 to 70.	Total.
Vertebræ	3	1	4	0	0	0	8
Hip	4	1	1	3	0	0	9
Knee	11	3	2	8	6	3	33
Ankle	2	0	3	0	2	0	7
Shoulder	1	2	2	1	0	0	6
Elbow	9	8	5	2	2	0	26
Wrist	1	3	2	1	3	0	10
Totals	31	18	19	15	13	3	99

Senile tuberculosis is very apt to be overlooked and to be mistaken for some much less serious condition, such as rheumatism or osteo-arthritis. Many of the cases, especially those involving the knee joint, demonstrate an important clinical fact, namely, that in its early stages this malady so closely mimics osteo-arthritis that the differential diagnosis is difficult; in fact, in the first few weeks it may be almost impossible. Senile tuberculosis commences with slight pain, stiffness, synovial swelling, limited effusion, and creaking of the synovial fringes on movement. The degree of muscular wasting corresponds to that usually present in osteo-arthritis. The presence of tuberculous disease of the lungs or the increasing thickening of the synovial membrane and the occurrence of night startings and loss of general health may furnish a clue to the true nature of the complaint. Usually doubt is soon removed by the onset of suppuration. I shall, therefore, offer some illustrations of the disease and relate my experience of two recent cases.

Case I.

J.M., a very debilitated man, aged eighty-three years, was admitted to the Lidcombe State Hospital on August 8, 1931, complaining of pain and swelling of the right arm of two months' duration. The right elbow, which was kept pronated and at an angle of about 130°, was 6·8 centimetres (two and three-quarter inches) larger in circumference than the left, and roughly globular in outline. The movements of the joint, which was somewhat painful and stiff, were restricted to a range of about 45°. The overlying skin was normal in appearance and was not hot. A large, soft, tender, fixed mass could be felt lying between the posterior aspect of the lower third of the humerus and the triceps muscle.

The heart sounds were distant and the apex beat lay in the fifth intercostal space, 9·3 centimetres (three and three-quarter inches) from the median plane. Crepitations were to be heard at the base of each lung. Nothing abnormal was detected in the alimentary or central nervous system. The temperature and the pulse and respiratory rates were normal.

The radiological report, dated August 8, 1931, read as follows:

> The lateral epicondyle, head of radius and upper portion of ulna are eroded; practically no new bone formation. I think this is probably a neoplasm.

When the patient was seen by me the following day, the following note was added:

> There is complete disorganization of the right elbow joint, with marked wasting of the muscles of forearm and upper arm. A soft swelling (? fluctuating) extends

[1] Read at a meeting of the members of the honorary staff of the New South Wales Community Hospital on March 3, 1932.

upwards behind the humerus for about a hand's breadth above the olecranon, to form a mass about the size of the palm of the hand. No sign of syringomyelia or tabo-paresis. Provisional diagnosis: senile tuberculous arthritis.

Exploratory puncture was performed, and the pathological report, dated August 28, states that tubercle bacilli were detected in the aspirated pus.

A further radiological report, dated August 24, was to the effect that there was no evidence of secondary deposits in the chest. At each apex there was a small area of mottling and fibrosis; these were regarded as tuberculous.

On September 2, 1931, amputation through the middle third of the upper arm was performed under "Avertin" anæsthesia. Equal antero-posterior flaps were used, and the muscles were divided by a circular incision. The patient's general condition gradually became weaker, and on the ninth day following the operation slight marginal necrosis of the skin flaps was noted.

On September 18 rupture of the entire wound occurred, exposing the unchanged sectioned tissues in which there was a complete absence of the usual reparative reaction. The patient succumbed four days later.

Case II.

T.P., aged sixty-six years, was admitted to the Lidcombe State Hospital on May 5, 1930, complaining of general debility and rheumatic pains in the back and legs. Stiffness and limitation of movement of the spine, especially in the lumbar region, were readily demonstrated, as well as slight limitation of movement and grating in both shoulder and both knee joints. Nothing abnormal was detected in the alimentary, circulatory or respiratory system. The blood did not react to the Wassermann test.

Under full diet, massage and a sodium salicylate mixture the patient improved sufficiently to be transferred in a month's time to the "yard" as a working patient.

Eighteen months later he complained of swelling of the right knee joint of ten days' duration. By November 25, 1931, the swelling had increased so that the outline of the synovial membrane was distinctly mapped out and fluctuation could be obtained in all axes of the joint. There was slight wasting of the thigh muscles. The skin over the joint was normal in appearance and was not hot. When seen in consultation on December 2, 1931, the right knee was much swollen and distorted, and measured 6·75 centimetres (two and a half inches) more in circumference than its fellow. The joint was flexed to 130° and the bones of the leg appeared to be displaced slightly backwards. The synovial cavity, distended with fluid, bulged on either side of the patella and for 8·0 centimetres (four inches) above it; but the swelling, instead of being firm and tense, was flaccid in character. On the lateral aspect of the joint the fluid had perforated the capsule to form a small hernia, or Baker's cyst, in the periarticular tissues. The synovial membrane was moderately thickened and its margin easily palpable.

The radiological report, dated December 8, 1931, read as follows:

> The articular margins of the bones forming the knee joint are eroded. The joint space is narrowed. The joint is not dislocated. The swelling of the joint is mostly in the soft tissues. The condition is probably due to septic arthritis. I do not think it is neoplastic. It is not like a Charcot's joint or gonococcal arthritis.

On the following day an attempt at aspiration and irrigation of the joint, by means of two needles inserted one on either side of the patella, failed, owing to blockage of the aspirating needle by flakes of lymph. A small incision was then made adjacent to the medial needle puncture and the joint cavity irrigated with saline solution. In all, about 240 mils (eight fluid ounces) of thin, greenish-yellow pus, containing many flakes of lymph, were removed. The incision was then sutured, circular pressure applied, and the limb slung in a Thomas splint with the knee joint slightly flexed and light weight tension applied. The pathological report, dated December 14, 1931, states that tubercle bacilli were detected in the aspirated fluid.

On December 13 the skin over the site of the hernial protrusion on the lateral aspect of the joint ulcerated and gave rise to a sinus. Subsequently sinuses formed on either side of the patella and the temperature rose and fell irregularly, varying between 36·1° and 38·3° C. (97° and 101° F.). The pulse rate varied between 80 and 101 beats per minute. The radiological report, dated February 3, 1931, read as follows:

> The joint space is now very narrow, due to destruction of all the cartilage; the upper ends of bone are also eroded. There appears to be progress in destruction since the last examination.

As the local mischief was increasing and the general condition of the patient becoming weaker, amputation was performed at the junction of the middle and lower thirds of the thigh on February 10, 1932; unequal antero-posterior flaps were used.

A further radiological examination performed on February 23, 1932, is to the following effect:

> The whole of the right lung is invaded by mottling and fibrosis. There is a similar condition on the left side down to the level of the third rib anteriorly. There are cavities at both apices. The condition is due to advanced bilateral tuberculosis, more marked on the right side.

Discussion.

The distinguishing features of the senile type of tuberculous joint might be enumerated as follows.

1. *The Course of the Disease.*—The course is more rapid and the process more destructive than in the young, and frequently, in so short a period as three months, suppuration and complete disorganization of the joint may have occurred.

2. *Relative Painlessness.*—In spite of the greater severity, the disease usually runs a less painful course.

3. *Intractability.*—It is well known that the prognosis in tuberculous disease of joints in patients over forty is very unfavourable. Suppuration is likely to occur, and the disease shows an obstinate tendency to advance. Indeed, recovery, when either the hip or the knee is attacked in patients between forty and eighty years of age, very rarely takes place.

4. *Mimicry of Other Joint Diseases.*—An axiom on which I have worked for many years is that when the clinical and radiological diagnoses are diametrically opposed, they are probably both wrong, and a fresh diagnosis should be sought. This axiom gave me the clue to the first case.

With regard to the second case, the first impression was that one was dealing with a large effusion associated with osteo-arthritis. Subsequently, until the pathologist solved the problem, the radiographic and operative findings strongly suggested pneumococcal arthritis. In the clinical examination more importance should have been paid to the thickening of the synovial membrane and less to the small amount of muscular wasting present.

In conclusion, it would appear that arrested pulmonary tuberculosis has the power in old age of setting up a tuberculous arthritis, which lesion runs a different and distinctive clinical course from that customarily observed in adolescence.

ACUTE DISSEMINATED ENCEPHALOMYELITIS WITH RECOVERY.

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History.

E.C., A SINGLE woman, aged fifty-six years, was admitted to the Royal North Shore Hospital of Sydney on November 21, 1931, under the care of Dr. F. Guy Griffiths. She related that eight days previously she had had severe frontal headache which had persisted for four days. During the

six days prior to admission there had been a sensation of "pins and needles" in the toes and fingers, also a trembling of the hands; and for three days "tight" pains around the shoulders and waist. She had not vomited. An indefinite influenzal attack had preceded the illness by two months.

Examination.

The patient had a fixed expression and slow speech, and was rather unemotional in spite of severe pains. An intention tremor was noted in both hands, and it was necessary to feed her.

The cranial nerves were not affected. Paresis of the muscles of both legs was present, especially the flexors of the thighs; there was no rigidity. There was loss of the sense of passive position and movement in the toes of both feet, with anæsthesia to touch over the cutaneous distribution of the fifth lumbar segment on the left side. The muscles of the calves were tender, but there was no cutaneous hyperæsthesia. The pupils were regular in shape, equal in size, and reacted to light and accommodation. The epigastric and abdominal reflexes were active; the knee jerk and ankle jerk on both sides were absent. No response was obtained when attempt was made to elicit the plantar reflexes, and no ankle or patellar clonus was present. The organic reflexes were not disturbed.

A mild degree of thickening of the radial arteries was noted. The systolic blood pressure was 155 and the diastolic pressure 90 millimetres of mercury.

Investigation.

Dr. E. Temple Smith, honorary ophthalmic surgeon, kindly saw the patient on two occasions, but found no abnormality in the optic fundi or fields of vision.

There were no abnormalities in the urine (until cystitis occurred on the twenty-third day). Radiological examination of the skull and sinuses revealed no abnormality. The cerebro-spinal fluid was clear, not under increased pressure, and did not react to the Wassermann test. Microscopical, bacteriological and chemical examination of the cerebro-spinal fluid revealed no abnormality. The blood contained 4,700,000 red cells per cubic millimetre; the hæmoglobin value was 70%; leucocytes numbered 14,000 per cubic millimetre (78% neutrophile cells). Each hundred cubic centimetres of blood contained 48 milligrammes of urea.

There was no reaction to Kahn's flocculation test nor to the blood Wassermann test before and after a provocative dose of "Novarsenobenzol".

Dr. H. C. McDouall, honorary psychiatrist, kindly saw the patient several times in consultation; he found no evidence of hysteria and agreed with the diagnosis of acute disseminated encephalomyelitis.

Course.

Throughout the illness the patient was drowsy; for a fortnight the general condition was poor and the outlook grave. She complained of tingling and numbness in the fingers and toes, and severe "tight gripping pains" in the back, which varied in position, being chiefly over the shoulders and thorax. A left facial paresis developed on the ninth day from the onset of the illness, but soon diminished, and had completely disappeared on the thirteenth day.

She gradually recovered, and on the thirtieth day there was no impairment of sensation nor paresis; the knee jerks were active and a flexor response was obtained on each side. She had also recovered from the cystitis.

The patient left hospital on the thirty-ninth day of her illness (December 29, 1931), having walked a few steps without assistance.

While she was in hospital her temperature remained within normal limits for the first ten days and afterwards was always below 37·8° C. (100° F.).

Dr. Guy Griffiths and I saw the patient in February and again in May of this year. She said that there had been no recurrence and that she had recovered completely except for slight weakness in the legs.

Examination revealed that she could walk well without support, and there was no defect of the nervous system.

Comment.

The lesions must have been scattered. Since there were no manifestations of syphilis elsewhere, no history suggestive of the disease, and none of its reactions, this disease was excluded. The patient was fifty-six years old, had no euphoria, no nystagmus, no scanning speech, no bitemporal pallor and no knee jerks, but severe pains; all these distinguish the disease from disseminated sclerosis. The slow onset, the order of advance, the subsidence and final recovery show that it was not Landry's acute ascending myelitis.

In this case the abrupt onset of the illness, the rapid and complete recovery of the patient, the weakness of the lower limbs, the ataxia of the upper limbs, the severe constricting pains, the paræsthesiæ, and the facial paresis, together with the absence of changes in the cerebro-spinal fluid make up MacAlpine's clinical picture of acute disseminated encephalomyelitis.

Summary.

1. A case with acute disseminated lesions of the nervous system is described, very similar to McAlpine's report of Flatau's series of seventeen cases, with no fever, no disturbance of organic reflexes and almost complete recovery.

2. Of interest was a transient facial paresis.

Acknowledgement.

I wish to express my thanks to Dr. F. Guy Griffiths, to whom I am indebted for permission to report the case.

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AN OPEN SAFETY PIN IN THE ŒSOPHAGUS OF AN INFANT FORTY-SEVEN DAYS OLD, REMOVED BY ENDOSCOPY.

By RAYMOND HENNESSY, M.B. (Melbourne), F.R.C.S. (England),

Melbourne.

IN these days of medical marvels it is not justifiable to report the removal of foreign bodies by endoscopy from the upper food passages and the air passages unless the circumstances are very unusual or a report is required for statistical purposes.

The writer ventures to report the following case on account of the tender age of the patient—an infant, 47 days old—from whose œsophagus he extracted an open safety pin. The pin was entirely within the œsophagus, that is, all parts of it were distal to the crico-pharyngeus muscle and not visible till this sphincter was opened by the beak of the œsophagoscope. The pin was open and lying vertically in the coronal plane, with the keeper and the point upwards and the spring downwards, the last being at about the level of the crossing of the left bronchus. The length of the keeper branch of the pin measured a shade over 2·5 centimetres (one inch), and the spread of the pin was 2·1 centimetres (five-sixths of an inch).

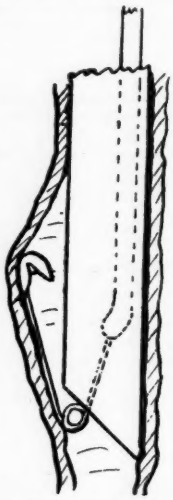
It is regretted that it is not possible to present an X ray photograph; but, unfortunately, the infant was

admitted to hospital after regular working hours, and the X ray technician was not available to take a film. The position and attitude of the pin, however, were very clearly seen and studied by means of the fluorescent screen.

The pin had been in the œsophagus for about six hours.

The instruments used were Jackson's special six-millimetre œsophagoscope for infants, and a light pair of Tucker's pin forceps. The operation was performed under light ether anæsthesia.

The method of removal was to ensheath the point branch of the pin by manœuvring it into the lumen of the œsophagoscope, then to grasp it with the forceps. The withdrawal was done with the keeper branch outside the lumen of the tube, the spring being held firmly against the lip of the distal end of the œsophagoscope. Thus the sharp point was adequately protected and could do no damage to the œsophagus during the extraction of the pin (see diagram).



The introduction of the œsophagoscope and the extraction of the pin occupied approximately three minutes.

Recovery was uneventful and the infant took the breast naturally soon after the operation.

Reviews.

DIPHTHERIA.

THE continued prevalence of diphtheria throughout the temperate zones has been for many years a matter for concern, and inquiry into the factors responsible is to be welcomed. In making an attempt to deal with these and kindred matters, Dr. J. Graham Forbes, Principal Assistant Medical Officer to the London County Council, has compiled an immense volume, the first half of which is almost entirely composed of statistics of the incidence and mortality of diphtheria in England and Wales, year by year, county by county, town by town—we had almost written, village by village. The title of the book, "Diphtheria, Past and Present, its Ætiology, Distribution, Transmission, and Prevention",[1] justifies us in expecting rather more than this; the greater portion of this section of the book presents itself as an unappetizing mass of indigestible figures. The maps of England and Wales and the accompanying tables and letterpress, showing the changes in geographical distribution of diphtheria during the past seventy years, may possess some local significance, but the general reader will be unable to detect it. He may gain a great deal of information, but is not likely to gain in knowledge or wisdom. In tracing the changes in prevalence over this period, Dr. Forbes is hampered by the fact that only for the past twenty years has notification been enforced, and he therefore has had to rely on mortality figures as a basis of mortality for the earlier decades. He has rightly added the deaths attributed to croup to those from diphtheria, and we find that undoubtedly the total mortality from diphtheria is very much less than it was forty or fifty years ago, but we know that a large number of cases reported nowadays would have been overlooked at that time, and a comparison of percentage mortalities becomes difficult. Dr. Forbes has been content to set down his vast array of figures almost without comment, and we could wish that he had collected within the scope of a score of pages all the essential statistics, together with any conclusions formed, and relegated the remainder to an appendix.

The reader will find with relief a short chapter on the means of transmission of diphtheria (pages 411-432). The portion dealing with the carrier problem is a veritable oasis, and we note with pleasure the increasing insistence on correction of pathological defects in the nose and throat as the *sine qua non* in the treatment of the carrier state.

The second half of the book will command more attention, being a survey of the progress of active immunization throughout the world. For a work of this size the details are surprisingly complete and up to date, and Dr. Forbes is to be congratulated on the completion of what must have been a very arduous labour. The work being done in Britain is described exhaustively, and there is particular interest in the attempts at immunization on a large scale in the Scottish towns of Edinburgh, Aberdeen and Dundee, and to a certain extent in London and provincial centres in England. The general conclusions are not new, but add convincing evidence of the value of immunization in the individual. It must be confessed that there is not yet sufficient evidence to enable us to assert that diphtheria can be permanently controlled in a large community; nevertheless the latest reports from New York and Canada indicate that if a sufficiently large percentage of the child population (possibly 40% or 50%) be immunized, there is every hope of a tremendous fall, both in mortality and incidence.

It is regrettable that in a work which is not a propagandist circular, but a collection of scientific data, there should be indications of bias. For instance, the gratuitous use of inverted commas in describing cases of "diphtheria" (*sic*) in the immunized is quite uncalled for. The cases referred to were cases of diphtheria; it is well known that such occur; some are fatal. This inexcusable departure from the dispassionate is indulged in frequently. Again, it is childish to refer to them as instances of "diphtheritic tonsillitis", as is done on page 726. The case for immunization is quite a good one; it needs neither excuses nor evasions.

To come nearer home, the following extract refers to recent conditions in Victoria: "A marked rise occurred in 1928 to a total of 4,100, following on the abrupt arrest of all immunization work after the Bundaberg events at the end of January, 2,700 cases being reported between April and September. In 1929 there was a decline to 3,055 cases, and in that year preventive inoculation was resumed in Melbourne and has been continued ever since. In the second and third quarters of 1930, 1,657 cases were recorded, 100 less than during the corresponding six months of 1929 and over 1,000 less than the same period of 1928."

The inference is plain that the rise in 1928 was due to the cessation of immunization following on the Bundaberg tragedy and that the fall in 1930 was due to its reinstitution in 1929. Such reasoning is not only faulty; it is dangerous. The antivivisectionist crank has just as much right to ascribe the very great prevalence of diphtheria in Victoria during the present year to the malign influence of immunization during the past two years as we have to ascribe its low incidence in 1930 to the benign influence of the immunization campaign of 1929. As a matter of fact, the total number of children immunized in Victoria during the past ten years has been too small to have any appreciable influence.

The extraordinary variations in virulence and malignity of diphtheria in Continental countries, notably Germany, France, Poland, Soviet Russia and the Balkans, make interesting reading. Here various modifications of the usual toxin-antitoxin and toxoid or anatoxin preparations, together with vaccines, have been tried, but the difficulties of dealing with an apathetic peasantry have in many districts hampered the work.

All over the world, however, where immunization has been tried on a reasonable scale, the recorded results are almost invariably favourable, and we are justified in regarding it as by far the best means of protecting any child from a serious attack of the disease. Dr. Forbes has done good service in bringing forward an immense mass of evidence in this direction, and it is to be hoped that the practice of immunization will be increasingly adopted, both by public authorities and by the family practitioner.

[1] "Diphtheria Past and Present: Its Ætiology, Distribution, Transmission and Prevention", by J. Graham Forbes, M.D., F.R.C.P., D.P.H., with introduction by Sir Frederick Andrewes, M.D., F.R.S.; 1932. London: John Bale, Sons and Danielsson, Limited. Demy 8vo., pp. 852, with charts. Price: 45s. net.

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THE MEDICAL PRACTITIONER AND MENTAL DISORDER.

In most uncivilized countries mental defectives and persons suffering from the various psychoses are either ignored or made sport of until they become a source of danger to the community, when they are either placed in durance or summarily dispatched. In some countries it is the custom simply to kill all insane persons, with the object merely of relieving the community of an incubus. Witch doctors and medicine men may be called in occasionally in the hope that they may rid the patient of the devil that has possessed him; but generally the treatment provided is negligible, the cruelty (often unwittingly imposed) is great, and the understanding is nil. In civilized countries procedures are more elaborate, though in some instances they are scarcely more intelligent. Within recent years much of the darkness obscuring the nature of mental disease has been dispersed, and there is reason for hope that the light of understanding may soon be cast on most of the problems it presents. In enlightened countries, then, the outlook for a person afflicted with a mental disorder is not so black as it was a few years ago. Still, there exists an air of mystery round the mental disorders, and in almost all sections of the community there is a certain superstitious wonder at the state of the afflicted. Mental disease is not generally regarded with the same cold logical scrutiny that is bestowed upon diseases of the physical being. It is time that the last relics of the superstitions that went with belief in afrits and witches should be shaken off and replaced by a scientific outlook. At present this scientific outlook is possessed by few, and the investigation of mental diseases is almost solely in the hands of the specialists. This is not as it should be. Progress must be slow unless the general practitioner takes the same interest in disorders of the mind as he takes in disorders of the body.

In the issues of this journal of January 23 and January 30, 1932, the Beattie-Smith Lectures for 1931 were published. These lectures, by Dr. John K. Adey, contain much valuable information and should be read by every medical practitioner. In the annual report of the Council of the British Medical Association, published in the supplement to *The British Medical Journal* (April 30, 1932), there appears a special report, entitled: "The Relationship of the Private Practitioner to the Treatment of Mental Disorder." Although this refers especially to the *Mental Treatment Act*, applicable only to England and Wales, it is worthy of close study by medical practitioners in Australia.

While knowledge has been increasing, the racial problems presented by the mental disorders have become more and more pressing. It is well known that many of the mental hospitals in various parts of Australia are taxed beyond their normal capacity. It is not proposed to discuss the reasons here. The mere fact that such a condition does exist should be sufficient to show that more interest must be taken by the great bulk of medical practitioners. The general practitioner has the nation's mental and physical health in his keeping; his duty to the community does not cease with the prevention and cure of bodily ills. He has greater opportunities for the prevention of insanity than any other member of the community; for he sees the beginnings of mental disease. Once recognizing the early symptoms, he may treat the patient himself, or, if he has not the time or inclination, or if he feels that he has not the necessary knowledge, he may

pass the patient on to a specialist. The patient's poverty is no hindrance to his receiving special treatment.

The tendency is often to handle impatiently the person suffering from a psychoneurosis, and to regard with awe or a feeling of helplessness the mentally deficient and the person afflicted with a psychosis. The impatience of a busy practitioner is readily understood, and perhaps sometimes excusable; but if he is too busy to be patient, he must allow the treatment to be continued by another. In these days there should be neither awe nor helplessness. Dr. Adey shows that much can be done by the general practitioner.

The general practitioner is very heavily laden; his responsibilities never cease to grow. He can be trusted to shoulder willingly this burden of responsibility in his relation to mental disorder. Doubtless he will continue forward whatever his load may be.

Current Comment.

THE SURGICAL INFECTIONS.

So much has been written on the subject of surgical infection since Lister first propounded his "germ theory", based on Pasteur's researches and discoveries, and so many side issues have been raised with regard to the question of immunity and treatment, that the time is indeed ripe for a general revision of theories and methods. To do this for all varieties of surgical infection would be a colossal task; but Stanislav Kostlivy has given a masterly exposition of the present-day position with regard to pyogenic and putrefactive infections.[1]

Invasion of the body by pyogenic organisms of sufficient virulence leads to the development of either a local or a general infection, depending on the result of the battle between the infecting organism and the body. Most writers ascribe only subsidiary importance to the virulence of the organism, and give pride of place to the resistance of the tissues, which depends on congenital or acquired biological attributes of immunity and the mechanism of disposal of organisms which enter the body. This question was recently discussed by Hedley Wright in THE MEDICAL JOURNAL OF AUSTRALIA; some of his views are incorporated.

In local infections there occurs a reaction in the tissues, consisting chiefly of hyperæmia, exudation, and, finally, a protective proliferation, with round cell infiltration, taking its origin from the reticulo-endothelial system.

The brunt of the attack against general infections is borne by the reticulo-endothelial system; thus in severe infections the liver and spleen degenerate. The part played by the bone marrow is an important one, the myeloid activity being manifested by an increase of neutrophile leucocytes.

Kostlivy utters a warning against attaching too great importance to morphological changes observed in response to infections, to the exclusion of disturbance of function. That the former are non-specific has been shown by experiences with protein shock therapy; but the modern advances made in colloidal biochemistry demonstrate how the tissue reactions may be altered and even the alkali reserve threatened in certain general infections, thus causing widespread disturbances of function. Although this local relative acidosis may be useful in combating the infection, the empirical rule of *"ubi pus, ibi evacua"* may be superseded. He advocates incision of areas of infection before the formation of pus, in order to free the body of foreign toxins, to release tension which promotes their absorption, and to cause a centrifugal flow of lymph and tissue fluids to the wound. The dangers of breaking through the defence barrier and opening up blood and lymph vessels are admitted, but the advantages of early incision are regarded as outweighing these risks, except in the treatment of anthrax pustules and the so-called malignant furuncles of the face, when conservatism is advocated.

The success which followed the introduction of quinine in the treatment of malaria and "Salvarsan" in syphilis, prompted the hope that similar selective substances would one day be found for other infections; meanwhile new antiseptics are continually making their appearance in large numbers, but their "index" determined *in vitro* becomes very much less *in vivo* on account of the defence barrier which must be penetrated before the infecting organisms can be reached. Antiseptics introduced into the blood stream act only by strengthening this wall, whilst those introduced locally can act only on germs within this barrier and cannot penetrate it. Thus there is little or no danger of absorption of antiseptics from abscess cavities packed with impregnated gauze; but signs of poisoning may occur if the same antiseptics are applied to indolent ulcers or reactionless wounds. Indeed, many workers claim that antiseptics act locally only by restoring the hydrogen ion concentration of the infected tissues to its optimum level. The position with regard to the *sterilisatio magna* of the entire body by chemotherapy as propounded by Ehrlich in the case of blood infections is even more in doubt. Any good results are probably due to stimulation and mobilization of non-specific defence reserves.

Next to the use of antiseptics, active and passive immunotherapy has to be considered in the attack against pyogenic infections. In the large majority of cases the active infection is overcome so rapidly

[1] *Wiener Medizinische Wochenschrift*, September 26, 1931.

that there has not been time for the formation of active immune antibodies, which can become established only in chronic sepsis. If the stimulus has not been great enough to produce sufficient specific antibodies, it may be helped by autogenous vaccines, provided the dose does not overload an already weakened body. Success so frequently follows rapidly upon the first or second injection of an autogenous vaccine, or not at all, that here again its effect is to be regarded as due to non-specific shock therapy rather than to an active immunity production. A similar explanation can be applied to the effects achieved by auto-pyotherapy and fixation abscesses.

The principle of passive immunotherapy is to produce antibodies against any known variety of bacteria, which can be introduced in the form of curative sera into a person lacking the particular antibody. No success has so far been attained in the production of antibody against the staphylococcus; with the streptococci each biological type has its own specific antibody that is useless against the other types; hence the so-called polyvalent antistreptococcal sera often fail; the same conditions apply to the pneumococci, but with more exactitude. Furthermore, the number of antitoxic units that can be introduced is small in relation to the large extent of surface from which the toxins can be liberated; and this may be an additional reason for the numerous failures encountered.

After dealing with Hedley Wright's and Besredka's views and non-specific protein shock therapy, Kostlivy goes on to discuss the methods of Orr and Albee. In 1924 Orr postulated certain basic conditions for the rational treatment of inflammation, with special reference to osteomyelitis. They were free incision, dependent drainage, protection against reinfection and mixed infection, increase of the natural resistance of the tissues, and maintenance of function. After a wide incision the wound was filled with vaseline and a complete unpadded plaster cast was applied and only changed if high temperature persisted or when it became soaked with foul pus. Albee extended this method to the treatment of purulent joint infections, and now it has been applied in the treatment of recent compound fractures and phlegmonous inflammations in soft parts, especially tendon sheaths. The focus of inflammation must be opened widely and washed out with physiological saline solution in all cases. All damaged tissues are excised and the cavity filled with sterile vaseline; a plaster cast is then applied to the whole limb. The subsequent course of the infection is amazing and the temperature usually falls by crisis, but the cast must not be prematurely removed if this does not occur. Thus, not only is the patient spared the pain of frequent dressings, but the work of the nursing staff and the expense are considerably reduced by these Orr-Albee methods; and Kostlivy urges their application in preference to the methods of continuous chlorine solution irrigations, with immobilization in splints, that have been in vogue since the Great War. At first sight these methods appear most unsurgical, and every modern surgeon must be horrified at the thought of shutting up, under an occlusive plaster cast, an inflamed and purulent wound without a tube and without even capillary drainage. But wound drainage only means an alteration in the flow of secretions from a centrifugal direction; and whether this be attained by drainage tubes, by capillary attraction, or by filling with vaseline; and whether the secretion escapes under a bandage or under the protecting cover of a plaster cast, the result is the same. The infrequent changes of dressing prevent the opening up of new pathways of absorption in the reaction barrier around the focus, and allow the wound to make full use of its own powers of defence. When the foul-smelling and pus-soaked plaster cast is removed, the limb is found to be bathed in a mixture of vaseline, clots of blood and pus; but the wounds are already covered with epithelium or filled with healthy granulations, and the surrounding skin is free of the signs of inflammation.

Kostlivy and Carsky have carried out some experimental work in an endeavour to arrive at an explanation of the efficiency of this treatment. Neither in animal inoculations nor in cultures did they detect any diminution in the virulence of the bacterial flora from the pus and discharge after it had collected under a plaster cast for weeks. Thus it is difficult to ascribe the results to the action of auto-toxins or bacteriophage. Local or general increased resistance of the tissues, due to immunization, was hardly a factor, because each premature change of dressing led to an immediate exacerbation of the inflammation. Hence the only theory tenable was that of a non-specific and local increase of tissue defence against the powers of invasion of the virulent infecting organisms, even anaerobes, which converted them into harmless saprophytes. The vaseline has a certain innate value; it envelops and inactivates the bacteria and either binds up or prevents the formation of their aggressins. But the more important factor is the absolute fixation of the entire limb. The importance of muscle movements in capillary blood and lymph flow has long been recognized. Pyogenic and anaerobic bacteria have no power of spontaneous movement and depend on passive movements for their invasion of fresh areas; if these movements are entirely prevented, or at least reduced to a minimum, the body can then deal with the bacteria *in situ*.

These methods have been successfully tried in Sydney by Graham and others for some time now; but in assessing the value of any method of treatment it must not be forgotten than man, although at the summit, is only a creature of Nature, and that Nature stands above all creations. In her view man may have no greater right to live than the bacteria; she is equally just or unjust to both. Man is inclined to consider everything from man's point of view and endeavour to apply the amphoteric laws of Nature to man's use, even to the extent of deceiving Nature; that he does not always succeed, or cannot at once succeed, is merely another of Nature's laws.

Abstracts from Current Medical Literature.

PÆDIATRICS.

"Epituberculosis."

J. C. SPENCE (*Archives of Disease in Childhood*, February, 1932) makes some observations upon the benign tuberculous infiltration of the lung known as "epituberculosis", which is dense and definite to all methods of examination, but clears up without leaving any pathological trace. Three cases are described, in each of which the skin reacted to tuberculin tests; no tubercle bacilli were recoverable from the sputum or stomach washings. In one case, however, on two separate occasions, tuberculous material was obtained by lung puncture. The condition is supposed by some to be of the nature of an allergic reaction around a tuberculous focus. Though the extent of the lesion may be massive, there is little disturbance of health beyond some slight pallor and loss of weight in the early stages. As regards Spence's two cases, in which no tubercle bacilli were actually recoverable, in one there was a history of contact with a tuberculous parent, neither patient had had pneumonia, and the complete disappearance of all signs was against the diagnosis of bronchiectasis or fibrosis. It is clear, therefore, says the author, that even a young infant has a great power of recovery from tuberculous lesions of the lung, a view that Armand-Delille and others have maintained for some years.

Rickets in Sydney.

KEMPSON MADDOX (*Archives of Disease in Childhood*, February, 1932) indicates the frequency of minor manifestations of rickets among children of the public hospital class in Sydney, in an examination (including radiological examination of the wrist in 129 cases) of 218 consecutive outpatient infants. Sufficient past or present clinical evidence, according to a specified standard, was found in 52%, the higher incidence falling between the ages of nine and twelve months. Radiographic signs of active rickets were found in 12·4%, and of healed rickets in 21%. With seven doubtful cases the total radiographic incidence amounted to 80%. On the whole, the disorder was of a mild to moderate degree and of an occult character. Marked beading and ligamentous laxity with abdominal and hepatic enlargement were most frequent in a small series of radiologically active patients. Spasmophilia was rare; craniotabes and epiphyseal enlargement were noticeably absent. Vitamin *A* and *D* deficiency could be anticipated in 43%. In 41 of 96 nursing mothers diet was deemed unsatisfactory from this standpoint. Breast-fed infants provided over a third of the radiologically active and half of the healed cases of rickets. Fresh milk from the country seemed to have a good protective effect, better at least than an insufficient amount of cod liver oil.

Ethmoiditis in Children.

P. SCHECHTER (*Archives of Pediatrics*, February, 1932) states that ethmoiditis is common in childhood, and that the symptoms may appear quite alarming, with a high temperature, nasal obstruction or discharge, and extreme prostration. The mucous membrane of the nose and throat is beefy red, often partly covered by muco-purulent discharge. The general symptoms may be associated with those of parenchymatous nephritis or gastro-enteritis. There is frequently a swelling at the inner canthus of the orbit, with marked tenderness to pressure over the ethmoid plate. The author is insistent on conservative treatment with adrenalin tampons, suction, and instillation of weak menthol solutions. In chronic cases treatment of enlarged tonsils and adenoids, which are present in 80% of cases, is essential. If surgical intervention becomes necessary, he prefers the external operation to the intranasal. Sinusitis in childhood is usually a pansinusitis.

Convalescent Serum in Whooping Cough.

J. C. S. BATTLEY (*Archives of Pediatrics*, October, 1931) used convalescent serum in two severe cases of pertussis (about 12 cubic centimetres given intramuscularly) and formed the impression that the treatment in one case was life-saving, and in both shortened the course of the disease. One child was a male infant, three weeks of age, when he contracted the disease. His brother, a twin, had already died of pertussis bronchopneumonia. His father was in the fifth week of the disease and provided the blood required. No vaccines were used, and for medication atropine solution (1 in 2,000) was given by mouth, and ether in olive oil rectally.

Vaccine Treatment of Asthma in Childhood.

A. G. MITCHELL AND M. L. COOPER (*Archives of Pediatrics*, December, 1931) confess that they have frequently failed with methods of specific protein desensitization. They discuss the various theories held as to the proper relationship of upper respiratory tract infection to asthma, and lean to the older view that, within limits, they are cause and effect, but state that vaccine treatment has had better success than local treatment to the nose. Their plan is to prepare a mixed autogenous vaccine from nasal swabbings obtained during an attack of infection of the upper part of the respiratory tract, according to the results obtained by skin testing with the organisms grown. One half to one minim forms the initial, and seven minims the maximum dose usually tolerated. The general results in a series of eleven children so treated were that they were mostly improved, especially when this treatment, which, they think, may be as much non-specific as specific, is combined with other recognized modes of treatment.

Studies in Juvenile Rheumatism.

CHRISTOPHER J. MCSWEENEY (*Archives of Disease in Childhood*, December, 1931) describes the results obtained from the inauguration of a rheumatism clinic in the city of Cardiff in 1927. The incidence of rheumatic heart disease was found to be six per thousand of the present school population. Every patient was seen at least once in six months, when he received a full medical examination, the numbers being limited to five or six new patients at each session. The main function of the clinic is, of course, educational. As regards the type of onset, 49% of 626 first attacks took the form of subacute rather than acute articular rheumatism, as compared with 48% in the London series. The majority of cases (66·3%) began in the winter or spring. This tendency was accentuated if the cases beginning with chorea were excluded. In 73·7% of instances the rheumatism began between the ages of five and ten years, again agreeing with the 1,700 London cases. The incidence of permanent heart disease since the inauguration of the supervisory scheme showed a fall from 60·2% to 23·3%, due to a scheme which insures the regular medical supervision of all rheumatic children and provides a special hospital for the treatment of early carditis. Almost 78% of the children under review did not come from the destitute class. Of the houses occupied by 600 rheumatic children, 61% were stated to be free of dampness. In a control group of non-rheumatic children investigated in 1927, the percentage of houses not damp was 62·2%. In 44·7% of family histories investigated, one or both parents of rheumatic children had themselves suffered during childhood from some form of acute rheumatism. As regards infectivity, the author concludes that while case-to-case infection in juvenile rheumatism is possible, it is neither the usual nor even a common mode of origin. In 6% of the cases investigated the onset of juvenile rheumatism was directly post-scarlatinal, and the author's experience has been that when rheumatism follows scarlet fever, it is less likely to be followed by grave cardiac sequelæ. No distinctive type of complexion or facies was recognized, apart from a preponderance of fair children with blue or grey eyes. The incidence of dental caries was slightly higher in the non-rheumatic group.

Normal Electrocardiograms in Childhood.

C. BRUCE PERRY (*Archives of Disease in Childhood*, October, 1931) has analysed the electrocardiograms of

fifty boys and fifty girls, selected by school doctors as having clinically normal hearts. All having a history of rheumatism and chorea were excluded. Boys were found to have a slightly lower cardiac rate than girls, and there was a slight tendency for the rate to fall with increasing age. Some arrhythmia (when the difference between longest and shortest pulse periods exceeded 0·1 second) occurred in 72% and was largely a factor of rate, not age. No other abnormalities of rhythm were detected. No changes occurred in the *P* wave, which was longest in lead II, smallest in lead III. The *P R* interval showed no constant variation with age or sex, though it was shorter than the average adult interval. The average in lead I was 0·142 second, in lead II, 0·148 second, and in lead III, 0·146 second. As regards the ventricular complex, the only change according to age was at five or six years. The author suggests that the change he observed represents the last remnants of the right ventricular preponderance of infancy. The author could not find the frequent absence or marked abbreviation of the *S T* interval recorded by Lincoln and Nicholson, the average duration being between 0·256 and 0·262 second, though the *S T* interval tended to be rather shorter in girls than in boys. The *T* wave in leads I and II was always upright and was inverted in lead III in 54% of cases. The electrical axis of the heart lay between 40° and 90° in 78% of cases.

ORTHOPÆDICS.

The Removal of the Meniscus of the Knee Joint.

OPINIONS were obtained from surgeons of ten countries (*The Journal of Bone and Joint Surgery*, April, 1932) in reply to the following questions: (i) Does the severity of the trauma bear any relation to the character and extent of the injury to the meniscus? (ii) In its removal, what incision do you consider advisable: the small lateral, the large lateral with section of the lateral ligament, the median patellar, the lateral patellar? (iii) Should the whole cartilage be removed or only the injured portion? With displaced, not fractured, cartilage, what proportion should be removed? (iv) What is the time of post-operative convalescence, in bed, before beginning weight-bearing on the leg or before allowing the full use of the knee? The majority of the surgeons considered that the severity of the trauma did not bear a relation to the extent of injury to the cartilage. The majority again expressed preference for a lateral incision with the occasional addition of the posterior incision. Opinions were evenly divided as between partial and entire removal of the meniscus. The period of stay in bed varied from three to fifteen days, with an average of about ten. Weight-bearing was first allowed after periods varying from three to twenty-eight days, with an average of about fourteen, and full use of the knee was allowed after periods varying from seven to fifty-six, with an average of about thirty days.

Vascular Diseases of the Extremities and Sympathectomy.

D. E. ROBERTSON (*The Journal of Bone and Joint Surgery*, January, 1932) discusses the causation of Raynaud's disease and describes methods of estimating the moistness of the skin. In his opinion the most prominent feature of a limb following sympathectomy is the dryness of the skin. He gives details of the results of sympathectomy in patients with Raynaud's disease, and concludes that: (i) In Raynaud's disease sympathectomy will relieve the symptoms of the disease and prevent the formation of gangrene. (ii) All cases of vascular diseases of the extremities have associated vasospasm in a varying degree; the vasospasm can be relieved by sympathectomy. (iii) Sympathectomy leaves no important deleterious effects, either in the lumbar or cervicothoracic excisions. (iv) Lumbar sympathectomy improves the bowel action.

Pain in Sprained Joints.

A. BRUNSCHWIG AND A. JUNG (*The Journal of Bone and Joint Surgery*, April, 1932) express the opinion that the pain in sprained joints is due primarily to trauma of the periarticular innervation, which is also responsible for the reflex muscular rigidity involving the injured joint. Periarticular injections of "Novocain" solution have a definite therapeutic value in the treatment of sprains, whereas interarticular injections have failed to relieve the pain.

The Treatment of Paralytic Talipes Calcaneus.

ROYAL WHITMAN (*The Journal of Bone and Joint Surgery*, April, 1932) compares the advantages of astragalectomy and backward displacement of the foot with tendon transplantation in the treatment of paralytic *talipes calcaneus*, and claims that the former procedure gives a more stable foot, and with one operation. He regards the restoration of the resistance or push of the foot as of far greater importance than voluntary balanced movement, and believes that it is indispensable to the stability of the limb when the anterior thigh muscles are involved.

Longitudinal Fractures of the Patella.

P. W. LAPIDUS (*The Journal of Bone and Joint Surgery*, April, 1932) describes the occurrence of longitudinal fractures of the patella in a vertical sagittal plane, and finds that they usually occur over the junction of the outer and middle thirds of the bone. This fracture is rarely recognized, but is not as uncommon as is usually believed. The clinical picture is clear: history of injury, localized linear tenderness over the lateral border of the patella, effusion in the knee joint, together with apparently negligible disability. Repeated aspiration of the knee joint is of value in treatment, and complete recovery may be expected within six to eight weeks, whereas lack of recognition of the condition may lead to a quite prolonged disability.

Fractures of the Neck of the Femur.

ELLIS JONES (*The Journal of Bone and Joint Surgery*, April, 1932) describes a new operative technique for treatment of fractures of the neck of the femur. After exposure of the trochanter and retraction of the *gluteus medius* muscle he removes a bone graft from the middle third of the lateral surface of the trochanter and inserts it through a hole drilled through the neck of the femur into the head. He claims that this procedure gives better exposure at the site of the fracture and is accompanied by more complete regeneration of bone than by the use of a tibial bone graft.

The Intervertebral Disks.

R. LERICHE AND A. JUNG (*La Presse Médicale*, April 18, 1931) discuss the mechanism concerned in the apparent compression of the intervertebral disk in certain diseases of the spine, particularly in Pott's disease. They describe the radiographic appearance, and regard the phenomenon as being due to compression of the disk owing to protective spasm of the muscles. The afferent impulse of the reflex originates in the nerve endings around the vertebral articulations and in the vertebral ligaments. They state that this apparent compression entirely disappears if the muscles in the vicinity of the disk concerned are infiltrated with local anæsthetic. A second radiograph after this procedure was apparently normal.

Spondylolisthesis.

R. H. BURNS (*Proceedings of the Royal Society of Medicine*, February, 1932) reports two cases of spondylolisthesis. One patient was a woman, aged twenty-eight years, who had first noticed a lump on the back during pregnancy. X ray examination revealed a gross forward displacement of the fifth lumbar vertebra. A fusion operation was performed, a stout graft from the tibia being employed. After three months on a plaster bed and one month walking in a support, the patient was able to resume normal activities. The second patient, a male, aged forty-eight years, had been pinned to the ground by a falling crane. He was kept in a recumbent position for six weeks, then allowed to walk while wearing a plaster jacket. A conspicuous feature in each case was pain in the calf of the leg. There was no history of injury in the first case.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Medical Society Hall, East Melbourne, on May 4, 1932, DR. B. M. SUTHERLAND, the President, in the chair.

Portrait of Dr. R. H. Todd.

A portrait of the late R. H. Todd was unveiled by the President, Dr. B. M. Sutherland. The unveiling was preceded by a short appreciative address by Dr. R. H. Fetherston, Dr. Todd's old schoolfellow and friend.

Angina Pectoris.

DR. M. D. SILBERBERG read a paper entitled: "Types of Angina Pectoris" and showed a series of lantern slides to illustrate his remarks (see page 35).

DR. J. F. MACKEDDIE congratulated Dr. Silberberg on the wealth of material drawn upon and the clear light brought to bear on a difficult subject. Dr. Mackeddie expressed approval of Huchard's positiveness on the question of classification. *Angina pectoris* was not only a symptom complex, but a definite disease with symptoms and pathology. Dr. Mackeddie deprecated the use of the term "pseudo-angina" and thought that positiveness in cardiac disease was associated with fewer mistakes than a more vague attitude. In regard to prognosis, Mackenzie put most emphasis on the ease of production of attacks. Dr. Mackeddie cited the case of an American traveller who had consulted him for symptoms of supposed indigestion. Dr. Mackeddie had considered he was suffering from anginal attacks, and advised immediate admission to hospital. He had been admitted to hospital and had died in a few days. Dr. Mackeddie also discussed the common association of gall-bladder disease and typical symptoms of *angina pectoris*.

DR. L. S. LATHAM said he had enjoyed the paper and had found himself trying to find parallels in his own experience in evaluating the symptoms and signs described by the lecturer. He agreed that careful investigation in each case and examination of the patient were essential, even in the trying circumstances so often surrounding an attack of acute angina. One should then take a definite stand as to diagnosis and treatment, difficult though that sometimes was. He agreed also that it was a pity to brand the neurotic patient as suffering from angina.

DR. W. OSTERMEYER expressed appreciation of the paper, and detailed some typical cases from his own experience. A patient with anginal symptoms also had hyperalgesia over the precordium following the attack. During examination fresh attacks had been precipitated on three successive occasions by touching this area. Dr. Ostermeyer considered that immobility during the attack was an important diagnostic sign. Some patients were breathless without actual pain, and breathlessness with immobility should always be considered suspicious of angina, even in the absence of pain. The therapeutic use of trinitrine was sometimes useful to distinguish between angina and coronary thrombosis. The use of strychnine, in his experience, was dangerous in angina, as he had seen death occur on two occasions within a few minutes of its injection.

DR. EWEN DOWNIE raised one point in connexion with coronary occlusion. Parsonett and Hyman, in a paper in the *Annals of Internal Medicine*, mentioned that there were 20% to 25% of diabetics in their series of cases of coronary thrombosis, and Dr. Downie had seen coronary thrombosis *post mortem* in a child of seven years who had died of untreated diabetes. With regard to insulin angina, which had been described by several authors, it had been found that diabetic patients sometimes suffered from an anginal type of pain two to four hours after insulin injections, and some had died later of coronary thrombosis. Dr. Downie asked for an explanation of this phenomenon.

DR. LESLIE E. HURLEY referred to the confusion which existed owing to the different terminology employed by various writers. An important point in diagnosis was to ask the patient what he did when the pain came on. The patient would frequently look surprised at so unnecessary a question, and would say, "I have to stop", or "I have to go slow", as though he felt the answer was obvious. *Post mortem*, no constant pathological changes were found in the hearts of patients dying with anginal symptoms. All the causative factors in angina were such as would raise the peripheral blood pressure, namely, excitement, cold, exertion, adrenal injections. On the other hand, the factors relieving angina, all had the reverse action—rest, warmth, nitrites *et cetera*. The suggestion was that anginal pain was due to increased cardio-aortic pressure in a heart which was the subject of organic disease. With regard to the association of angina with diabetes, Dr. Hurley thought that elderly diabetics could possibly be harmed by too rigid treatment by diet and insulin; as their cardiac musculature had become used to a raised blood sugar content.

DR. W. J. BEVERIDGE described a hospital patient whom he had seen in Western Australia, an elderly man, who had had attacks of anginal pain that had been so severe as to have been relieved only by morphine. He had been seen and examined by numerous physicians, who had made various diagnoses, varying from malingering to coronary disease. The man later had returned to his farm and had been seen by a local medical practitioner in an epileptiform fit; his condition had then been diagnosed as epilepsy. From that time definite epileptic attacks had alternated with or had been initiated by cardiac pain.

DR. B. M. SUTHERLAND expressed the thanks and appreciation of the meeting for Dr. Silberberg's paper.

Dr. Silberberg, in reply, said that in dealing with patients suffering from anginal symptoms, one was often at a loss to know what was the true nature of the pain. He considered that Sir James Mackenzie's work on *angina pectoris* was a mine of information, wonderfully put together. Coronary thrombosis had not been singled out as a separate clinical entity, even by such eminent observers as Osler and Wenkebach. As an illustration of the mixed type of angina Dr. Silberberg cited the case of a woman, living in the western district, who had suffered from severe anginal symptoms at about the time of the menopause. She had had a high blood pressure (about 200 millimetres of mercury, systolic), and her heart was enlarged, the apex beat being 13·75 centimetres (five and a half inches) to the left of the middle line. Her husband had died when she was about forty years old, and she had been forced to work very hard to support her family and to undertake considerable responsibility. She had been treated by a number of different physicians, and her pain had been relieved only by injections of morphine. Later it had been found that injections of water also gave her relief, and it had become obvious that there was a neurotic element in her condition. Her trouble, however, had recurred; but at the age of about fifty-seven she had obtained great help from reading a book on the "psychology of the soul" and was still working hard and efficiently. Referring to the well known relation between angina and gall-bladder disease, Dr. Silberberg remarked that one point in common was that they belonged to the same age group, but their association in many cases seemed to be more definite than this, and it was doubtful whether the anginal pain was due to a toxic action on the heart muscle or to a reflex nervous action. In coming to an opinion about the prognosis in any case of angina, the medical practitioner must consider the presence of painful dyspnœa as a dangerous symptom. Anrep's work showed that there were vaso-constrictor nerves to the coronary arteries, and therefore, that coronary spasm was a possibility. It was also possible that the *musculus triangularis sterni* might go into spasm and be the cause of the feeling of constriction in some cases. In patients who during life had shown signs of unexplained pericarditis, *post mortem* examination in some cases had revealed coronary infarcts, and it was possible that in other cases pericarditis might be due to coronary thrombosis of a degree not sufficient to cause death. Possibly the explanation of anginal symptoms following injections of insulin was the sudden hypoglycæmia due to the insulin. This might be comparable to the less

intense sensation of faintness or sinking feeling which occurred in normal people when a meal was missed on a strenuous day. This sensation of weakness might be due to a spontaneous hypoglycæmia. Referring to Dr. Hurley's remarks on peripheral blood pressure, Dr. Silberberg said that Lewis had complained that too few observations had been carried out on the pulse and blood pressure during actual attacks of angina. Under his (Lewis's) guidance such an investigation had been carried out in London, and it had been found that attacks of angina were not necessarily coincident with rises in blood pressure, and that relief by amyl nitrite was not coincident with lowering of blood pressure. This tended to indicate that amyl nitrite acted as a coronary dilator rather than in lowering the general blood pressure. Finally, Dr. Silberberg said that congestive heart failure did not invariably relieve anginal pain. He thanked members for their reception of his paper and for the very interesting discussion which had followed.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Darling Building, the University of Adelaide, on April 28, 1932, DR. A. BENSON, the President, in the chair.

Cardio-Vascular Syphilis.

DR. J. B. CLELAND demonstrated specimens of cardio-vascular syphilis and read a paper entitled: "Cardio-Vascular Syphilis as Revealed at Post Mortem Examinations" (see page 46).

DR. GUY LENDON read a paper entitled: "Syphilis of the Heart and Great Vessels" (see page 41).

Anæsthetic Apparatus.

DR. C. R. E. DOWNING demonstrated an anæsthetic apparatus of his own devising—the turbine suction ether apparatus. Dr. Downing remarked that the outstanding feature of the apparatus was that, instead of several working parts (an electric motor, a two-way pump, the heating element for the ether bottle), all three functions were performed by the motor itself. The principle employed was that embodied in one of the best known types of domestic cleaner. The air was sucked in by the high speed turbine at one end and expelled at the other; thus there was no necessity for a pump. The air was heated by the motor and was then ready to be passed directly through the ether; thus the necessity for a separate heating unit was done away with. The whole apparatus could be assembled in one or two minutes. Blood could not be sucked into the pump and cause trouble, for the motor was enclosed. The motor ran at a constant speed, causing an even suction. For the additional sum of £4 or £5 the type of vacuum cleaner utilized could be adapted entirely and successfully, without in any way altering or interfering with the function of the cleaner as such, and all that was required to convert the domestic machine into a surgical apparatus was the removal of one vulcanite screw-piece from the suction end and replacement by another. Likewise, a replica end-piece was placed in the exhaust end of the machine to convey the heated air to the ether bottle. To adapt the machine for the endotracheal administration of ether, it was only necessary to replace the small tube at the release valve by a tube of sufficient length to reach from machine to patient.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal Prince Alfred Hospital, Sydney, on May 12, 1932. The meeting took the form of a series of clinical demonstrations by members of the honorary staff.

Asthma.

DR. ROBERT S. STEEL showed a number of patients with asthma who were undergoing treatment. Positive skin tests were demonstrated, and also the technique employed in this type of investigation.

On account of the frequency of sensitivity to house dust in the city, six patients sensitive to house dust were selected. In three patients house dust sensitivity occurred as a single manifestation. Of these two were under treatment with house dust extract prepared by the Commonwealth Serum Laboratories, and the other was being treated by an extract prepared from her own house dust by Dr. Durie. All three were decidedly improved, as evidenced by decreased severity and number of the attacks of asthma, and, in one case, relief. Dr. Steel remarked that these patients showed the marked benefit usual in persons sensitive to but one allergen, who had no associated infection. Another patient was shown to be sensitive to horse dander and Kentucky blue grass as well as house dust; the result of treatment by desensitization was pronounced.

Two patients sensitive to house dust, and suffering from associated bronchitis, with some degree of fibrosis of the lungs, were also demonstrated. Both of these had derived considerable benefit from treatment with house dust extract, and were receiving injections of autogenous vaccine prepared from the sputum, with increasing benefit. Dr. Steel remarked that these patients illustrated the well known fact that infection, in the nature of a chronic bronchitis, will prevent the house dust extract (or any other extracted allergen) from bringing about complete relief of symptoms to allergic patients.

A man, aged seventy-five years, was shown, to whom an autogenous vaccine had been administered for about eight months, with complete relief of his asthma. Dr. Steel said that this case illustrated the prolonged treatment and care in dosage which was considered advisable in bacterin-sensitive (bronchitic) individuals. Injections were given weekly; great caution had to be exercised in increasing the dosage.

Dr. Steel's last patient was a baker who only suffered from asthmatic attacks in the bakehouse. Positive skin tests were shown in the case of flour, rice, rye and volymost. An extract was prepared from materials used in the bakery and an attempt was in progress to desensitize him with the extract.

Dr. Steel pointed out that all the asthmatic patients were taught to do breathing exercises, consisting of passive slow expiration and rapid inspiration.

Addison's Disease.

DR. ALLAN S. WALKER showed a woman, aged forty-two years, who complained of weakness that had existed for two years. There was nothing of importance in the family history, and the patient had had no previous illness. She had noticed for seven years that her skin was becoming darker; but this change had advanced rapidly during the previous two months. She felt disinclined for any exertion. She had fainted several times during the two months before admission. For six weeks before admission she had suffered from increasing nausea and had vomited every day. She had no special pain except, since coming into hospital, in the right temporo-mandibular joint, which was swollen and sore. Examination revealed the most intense pigmentation, more pronounced on the exposed parts and the parts subjected to pressure. The characteristic brown line on the lips was present. A few moles had taken on the most intense black colour. The abdomen was stippled with pigment in parts, and there were interspersed areas of vitiligo. There was nothing abnormal to be found in the organs of the thorax or abdomen on usual examination. Loss of weight was not very pronounced before, but had been considerable recently, as she had hardly been able to take any food on account of the nausea or vomiting, which had not responded to any medication. The systolic blood pressure was 70 and the diastolic 50 millimetres of mercury at the time of admission. The reading was difficult to take, whatever method was used. The blood cholesterol content was 60 milligrammes per hundred cubic centimetres.

Radiological examination of the chest revealed no abnormality, and of the abdomen some thickening of the gall-bladder with a few gall-stones, but nothing suggesting areas of calcification in the suprarenal region. The white

blood cells numbered 10,000 per cubic millimetre. The administration of adrenalin produced no change. Some whole gland adrenal extract was given by mouth, with apparently no effect, save to make the patient complain of nausea. Glucose was given in saline solution *per rectum* when she was unable to retain food. Her condition fluctuated considerably, with no definite cause, though the general tendency was towards deterioration.

Inquiry was made with a view to obtaining extract of adrenal cortex, to be given by injection, as very favourable results had been reported from other parts of the world. This hormone was not available.

Dr. Walker remarked that this patient conformed most strikingly to Addison's classical description.

Lymphosarcoma.

Dr. Walker also showed a child, aged four and a half years, who, ten weeks previously, had an attack of abdominal pain and vomited. After that time she lost weight and grew pale and weak. Seen about six weeks before the meeting, she was found to have a large liver and spleen, and there was a definite secondary anæmia, the red blood cells numbering less than three million per cubic millimetre. The picture then suggested splenic anæmia. Lymphadenoma was also thought of. About two weeks before the meeting she was admitted to the Royal Prince Alfred Hospital looking obviously ill and much changed. The liver was huge, the spleen could be felt, and there were masses of discrete glands in the left posterior triangle of the neck. A blood count revealed only 1,800,000 red blood cells per cubic millimetre of blood. Transfusion of blood was carried out, and a gland was excised from the neck at the same time. The red blood cells rose in number to 2,700,000 per cubic millimetre, and the hæmoglobin content from 20% to 30%. The white blood cells increased in number from 4,000 to 6,000 per cubic millimetre. Monocytes were noted to be in the proportion of 13% of the white cells, the neutrophile cells 48%. There were 10% of metamyelocytes. The count did not preclude the possibility of a blood dyscrasia.

The lymphatic gland removed was very soft and red. Section revealed a histology very different from that of lymphadenoma or a blood disease. The cells appeared of an anaplastic type and were thought to suggest a growth of sarcomatous type arising from lymphatic or reticular cells. The appearance was not unlike some of the tumours described as arising from the adrenal, namely, neuroblastoma. Some nodules could now be detected on the skull, suggesting metastases, such as occur in bones in this condition, and radiological examination was to be made forthwith.

Deep radiation was commenced, but had effected little visible change at the time of the meeting.

Primary Suture of Muscles and Nerves Followed by Gas Gangrene.

DR. J. COLVIN STOREY showed a male patient, aged eighteen years, who had been admitted to the Royal Prince Alfred Hospital on September 14, 1931, a few hours after having fallen into a window and thereby severely wounded his left forearm just below the elbow joint. The lad's father had controlled the bleeding at once by firmly grasping the forearm with both hands so as to encircle it. The patient was brought to the operating theatre with a tourniquet applied, rather pale from loss of blood. Under ether anæsthesia the wound was examined and washed with ether, and some pieces of glass were removed. The following structures had been divided: the common flexor muscles, the median nerve, the ulnar artery, the radial artery, the biceps tendon (immediately above the insertion into the bicipital tubercle), the supinator muscle, the large muscular branch of the radial nerve, part of the *extensor carpi radialis*, numerous branches of arteries. The veins corresponding to various arteries had also been severed. The injured arteries and veins were ligated. The median nerve was sutured with fine silk; the cut branch of the radial nerve was sutured. Muscles were sutured with plain catgut. Difficulty was experienced with the biceps tendon, and finally a clove hitch of thick catgut was put round the stump of insertion and the ends sutured to biceps and brachialis. The skin was lightly sutured with silkworm gut, after the whole wound had been smeared with bismuth iodoform and paraffin paste. Gas gangrene antiserum in a dose of 10,000 units was given intramuscularly, together with 1,500 units of tetanus antiserum.

On September 17, 1931, bubbles of gas appeared at the outer end of the wound; 10,000 units of gas gangrene antiserum were then injected. On September 18, 1931, pus and gas, with a foul odour, came from the outer end of the wound. A smear and a culture were made; *Bacillus welchii* was reported to be present. Gas gangrene antiserum in a dose of 10,000 units was given. On September 19, 1931, pus and gas were still present in the outer end of the wound; 10,000 units of gas gangrene antiserum were given. On September 28, 1931, the patient was very well. On October 3, 1931, a splint was applied to the forearm. By October 13, 1931, the patient was fit to be discharged, to return for dressings.

The temperature rose to 39·2° C. (102·6° F.) on August 15, 1931, and became normal by September 27, 1931.

Dr. Storey remarked that this patient had reported once a week for some time after leaving hospital. The limb had been kept in a sling, with a light aluminium splint to the forearm for the first few weeks. After the first fortnight voluntary movement at the elbow, only so far as it could be obtained without pain, had been encouraged. No passive movement or massage had been employed. On tapping over the course of the median nerve, feelings of "pins and needles" had been elicited from a point which had moved progressively down the forearm. Feeling had returned in the distribution of the median nerve one month before the meeting. Most of the intrinsic muscles of the hand had wasted, and it had been thought that there was in this patient an anomalous distribution of fibres of the median and ulnar nerves. There had been no anæsthesia in the area supplied by the ulnar nerve. It could now be seen that there was full voluntary flexion at the elbow, and full supination, whilst extension was still not quite complete. The scar was now supple. The course of the infection, with its happy termination, had been *prima facie* evidence of the efficiency of gas gangrene antiserum. The patient had never looked very ill, and only the outer part of the wound had apparently been affected.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Hagger, Thomas Dudley, M.B., B.S., 1930 (Univ. Melbourne), Children's Hospital, Carlton, N.3.

Lawson, Robert Sutherland, M.B., B.S., 1931 (Univ. Melbourne), Melbourne Hospital, Melbourne, C.1.

Correspondence.

ERYTHRŒDEMA.

SIR: I have had to treat several children suffering from erythrœdema and they have all done well when their milk feedings have been replaced by a mixed diet rich in protein. Other treatment, except for the administration of "Ostelin", has been merely symptomatic.

The last baby I treated was brought to me on April 25, 1932. He was seven months old, had been breast fed entirely for three months, then for the two next months the breast feeds had been supplemented with "Lactogen". The last two months he had had cow's milk and water in equal parts.

He had been very cross for a month, and on examination showed all the typical signs of the disease. His bottle feeds were reduced to two a day and were composed of cow's milk, one part, to two parts of water. For his other meals he had eggs, brains, finely chopped liver and steak,

all young vegetables, mashed potatoes, stewed fruit, raw apples and pears, and a small amount of farinaceous foods.

He improved remarkably quickly, and the diet did not upset his digestion. In seven weeks' time he was quite well, the photophobia being the last symptom to disappear.

Yours, etc.,

E. KENT HUGHES.

Armidale,
New South Wales,
June 16, 1932.

ANTHRAX.

SIR: In Dr. Ian Hamilton's excellent case report of anthrax (malignant pustule) in the current week's issue of the journal, he comments that no Australian case has been reported since Cleland's in 1923.

May I draw his attention to a report of a case of malignant pustule treated without excision at the Newcastle Hospital, New South Wales, and reported in THE MEDICAL JOURNAL OF AUSTRALIA on February 21, 1925?

In this case also infection was conveyed by the bristles of a cheap shaving brush.

Yours, etc.,

THOMAS HAMILTON.

Newcastle,
New South Wales,
June 20, 1932.

THE WORKERS' COMPENSATION ACT.

SIR: When certain insurance policies under the abovementioned Act were being checked, a renewal form was found to have the following notice attached.

MEDICAL ATTENTION.

Unless injured employees are unable to travel, they should be sent as soon as practicable after the happening of the accident to our office, Manufacturers' House, 26 O'Connell Street, Sydney, where we have our own fully equipped casualty clinic for their treatment. We have two doctors in attendance from 9 a.m. to 5 p.m. Mondays to Fridays, and 9 a.m. to 12 noon on Saturdays.

We have an up-to-date X ray plant with an expert radiographer in attendance during the hours stated above. Our medical advisors can obtain an X ray of any injury within ten minutes of the patient arriving, thus being in the position to correctly diagnose the case.

A trained nurse and fully qualified masseurs are always in attendance to give the treatment ordered by our doctors. For this purpose we have installed special and expensive apparatus, and we are able to promote complete recovery in the shortest possible time. Hot air, diathermy, ionization, faradism and other varieties of electro-medical therapy are always available.

Medical accounts for treatment given elsewhere must not be paid without our written authority, in accordance with the provisions of the *Workers' Compensation Act*. (See page 77, Appendix, Condition 3.)

We invite our clients to inspect our medical clinic at any time. Our Company is the only licensed insurer which has established a clinic within its own premises for the exclusive treatment of your injured workers.

Please impress on your employees the advantage of obtaining treatment by our doctors.

Manufacturers' Mutual Insurance Ltd.
26 O'Connell Street, Sydney.

Apart from the disadvantageous position (in the event of litigation) in which an injured worker would be placed as regards obtaining medical evidence as to the clinical course of the sustained injury or disease, oblique advertising of the above nature would induce an employer to be a tool in inadvertently endeavouring to sever the connexion between the injured worker and his customary medical attendant, and cause him to refer such employee to the said insurance company.

Such a procedure would appear to be at variance with the workings of the *Workers' Compensation Act* as enunciated by the Honorary Medical Secretary of the New South Wales Branch of the British Medical Association and published in THE MEDICAL JOURNAL OF AUSTRALIA on February 14, 1931.

Yours, etc.,

H. RUTHERFORD DARLING.

229, Macquarie Street,
Sydney,
June 25, 1932.

PHYSICS.

SIR: I have always taken an interest in physics, and frequently read the subject, obtaining knowledge and pleasure from doing so; but I am afraid that I met my Waterloo when, recently, I attempted to follow the physics as taught to the first year medical student.

Surely the time for reform of this course in the medical curriculum is long overdue.

Owing to the neglect of this subject by the medical members of the faculty, it has been left to the physicist (who, like most specialists, considers his subject of paramount importance) to elaborate and enlarge this subject, which to the student is only an ancillary one.

The present day text book is a wonderful example of the printer's and bookmaker's art (Australasian Medical Publishing Company, Limited) and a monument to the knowledge and application of its authors (Booth and Nicol), but as an elementary text book for medical students it is as useful and interesting as a railway time-table.

All that the medical student needs is a grounding in the properties of matter, heat, light, magnetism and electricity, as applied in medicine. The text book, however, is crammed from beginning to end with abstruse formulæ couched in the alphabets of England, Greece, Arabia and some unknown tongues, probably the Indian sign language or Mah Jongg.

Nearly two-thirds of the book consist of unadulterated mechanics and higher mathematics which are in no way necessary in considering elementary physics.

The medical students form the majority of fee-payers in the first year, and they should be served with a course of real elements, and the frills left to the faculties of science and engineering in separate classes.

In the days of Threlfall and Pollock a simple and understandable course was given, and such a course should be reverted to and the present course left for the mathematical prodigies and the budding Einsteins.

Yours, etc.,

J. G. EDWARDS.

185, Macquarie Street,
Sydney,
June 22, 1932.

"AVERTIN."

SIR: May I voice my approval of your recent leader on "Avertin" anæsthesia? I sometimes wonder to myself why "Avertin" is ever used. Amongst all basal anæsthetics it is under the gravest suspicion.

Sydney is the fourth city in which I have found uneasiness about the safety of this drug and whispers about fatalities. In Vienna, London and Manchester I found eminent men afraid to use it. On looking over the abstracts of recent medical literature, I repeatedly find the same warnings.

Schröder believes the combination with "Narcelyn" to be "ideal", the "Narcelyn" going far to combat the toxic effects of "Avertin", the net result being a mortality of only 3%, and this in gynæcological operations.

Stark reviews the statistics to date and finds the mortality at least as high as in chloroform anæsthesia. And

so on. The very best that I can find about "Avertin" is in Dr. Maddox's book—a mortality of three in 10,000, when all allowance is made for operative risks.

With open ether a few years ago there were no anæsthetic deaths in three years in one of our metropolitan hospitals, a mortality of none in 11,260, all operative risks included. And so it will not suffice to quote one or two hundred cases of "Avertin" anæsthesia. Even if "Avertin" increases the mortality by only three in 10,000, it should be some consideration. The drug seems to have no redeeming feature. It cannot even be credited with convenience of administration.

Is it not advisable to give up "Avertin" without waiting to prove the mortality in our own experience?

On the other hand, compare the reports in the literature on the barbiturates—the recent tributes by Sir Harold Gillis and Cecil Rowntree, of the Cancer Hospital, and the recent reports by Lundy and Kast, abstracted in *The British Medical Journal*.

What an excellent lesson there is in the recent address by Dr. Julian Smith! "I can recall a series of six or seven operations that I did with delightful ease, but at that particular time one heard here and there of a disaster, and so I just quietly refrained from using any more." The words were not used of "Avertin", but how aptly they may be applied, and how profitably they may be pondered!

Yours, etc.,

VICTOR J. KINSELLA.

171, Macquarie Street,
Sydney,
June 25, 1932.

Proceedings of the Australian Medical Boards.

VICTORIA.

THE undermentioned has been registered, pursuant to the provisions of the *Medical Act*, 1928, of Victoria, as a duly qualified medical practitioner:

Storer, Robert Vivian, M.R.C.S. (England), L.R.C.P. (London), 1923, 167, Collins Street, Melbourne.

Additional degrees and diplomas registered:

Burke, Frank David, M.D. (Melbourne), 1932.
Tymms, Eric Mortimer, M.D. (Melbourne), 1932.
Duncan, Lois Madge, D.P.H. (Melbourne), 1932.
Mitchell, Douglas Harold, F.R.C.S. (England), 1931.

Books Received.

CLIO MEDICA: A SERIES OF PRIMERS ON THE HISTORY OF MEDICINE, Edited by E. B. Krumbhaar, M.D.; Volume VI: Italian Medicine; 1932. New York: Paul B. Hoeber. Foolscap 8vo., pp. 134, with illustrations.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

LAUNCESTON PUBLIC HOSPITAL, TASMANIA: Resident Medical Officer (male).

MANLY DISTRICT HOSPITAL, SYDNEY, NEW SOUTH WALES: Resident Medical Officer.

TOOWOOMBA HOSPITALS BOARD, QUEENSLAND: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

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